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THE
Diseases of the Newborn

BY

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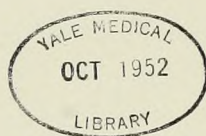
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Author's Preface

It is usual to divide the subject of pædiatrics, which comprises human physiology and pathology from the first day of life until the completion of sexual maturity, into several age-periods according to the various stages passed through in development. There is no desire to set up different and independent subjects by this method of grouping; it is more for the sake of clearness of arrangement. Moreover it is a logical result of the differences in the bodily and mental development and the general dietetic and hygienic requirements during each of these epochs; and also it follows from the differences in the nature, appearance and course of the pathological processes.

The period of infancy, which is taken to terminate at the close of the first year of life, has had the first days and weeks of life set apart, quite rightly, into a special division—the newborn period of infancy. As the different stages of childhood are not sharply defined but rather tend to pass imperceptibly one into the other, so this newborn period passes gradually into the rest of infancy. Théoretically the term includes that period of time in which the vital processes are still under the influence of those violent changes involved by the abrupt transition from intra-uterine to extra-uterine life. The processes occurring in foetal life continue to hold sway in that period immediately following birth; the intestinal mode of nutrition comes at first by degrees into working order; the child reacts to the stimulation of the outer world, to the influence of which it is for the first time exposed, in a different manner to what it does later, and the metabolic processes show certain differences compared with those of the older infant. The peculiarities of the newborn state are most pronounced in the first days after birth; they diminish by degrees when after two or three days of loss of weight the child again continues to gain flesh, and nourishment from the mother's breast comes into full play; it then needs a certain time before the organism, after the abrupt change of all its vital processes, pursues an orderly course. The result of all this is that a number of diseases are peculiar to the newborn child which are not met with in later life—not, at least, in the same form—and they make up for those diseases that do not appear at this early age.

How long shall a child be regarded as "newborn"? One has sought to find outward signs for marking off this period of life,

and most often the day upon which the umbilical stump has fallen off is taken as its termination. But as the separation of this necrotic remnant has no effect on physiological processes and the time of its occurrence varies considerably, it can hardly be taken as a basis for defining the period. Moreover it has to be taken into account that there are diseases arising from the navel which are generally regarded as diseases of the newborn; diseases which extend beyond the time of the separation of the umbilical stump. A more physiological standard, inasmuch as it refers to internal processes, is the course of the weight curve. The weight curve of the foetus and the infant does not show a continuously ascending line. The sudden changes of the vital processes at the moment of birth cause a reaction which in many respects involves a delay in the ensuing development. The feeding of the child during the first few days has for its aim the making up of loss; from the moment of birth to the time when the birth weight has been regained one finds that there is no true increase but rather a replacement of the material consumed, and particularly water, which the organism has lost in the first few days of life. The first ascent of the weight curve has therefore an essentially different significance to the increase which indicates growth which occurs after the body weight has returned to its starting point. One cannot without justification take this day as the end of the newborn period. It occurs under ideal conditions about the middle of the second week; repeatedly, however, at an earlier or a later time, so that one cannot draw a sharp line by this means. As regards the employment of a wider outward sign, namely, the breast secretion, although one deals here with a symptom, which is absolutely characteristic of this early period, it is unsuitable because the secretion is wont to last to a time when one can hardly regard the child as newborn. Equally variable are those limits that may be furnished by the duration of icterus neonatorum. And the course of those true diseases of the newborn that commence at this early stage gives no better standard. On general grounds, therefore, one would lay down the first two or three weeks as the newborn period, with the reservation that many children lose early the clinical characteristics of the newborn state, while in others these peculiarities persist for a longer time.

While it appears justifiable on clinical grounds to deal with the newborn period separately, it is equally desirable to do so for practical reasons also. We find ourselves here on the border-line between pædiatrists and obstetricians, and it is often debated whether the treatment of the newborn and the management of its feeding should be the duty of the accoucheur or the pædiatrist. It is quite obvious that the scope of the specialist in diseases of children should include the whole of childhood, and the first few weeks of life can be made no exception. On the other hand, it cannot be denied that the care of the newborn often falls quite as much on the medical attendant who undertakes the delivery of the

mother. The child is very liable to suffer as the result of this border-line conflict. Thus the specialist in diseases of children mostly fails to have the opportunity to acquire a thorough knowledge of the newborn child, and many obstetricians fail to utilize fully those opportunities, because the study of the child has not much to do with their speciality. A result of this is that in most books on diseases of children the newborn child is discussed under the old heading of "diseases of the newborn," and the practically important side of this period, the technique of nutrition, is only dealt with quite cursorily, while many textbooks on obstetrics have absolutely nothing to say on the physiology and pathology of the child, or at least only in an incomplete and out-of-date fashion.

To the question, On whom does the care of the newborn lie? there is really only one answer: On the specialist. Whether he is an obstetrician or a pædiatrist is then of no consequence. But just as little should the knowledge of an infants' doctor be confined to the period of infancy as a "newborn specialist's" to the lying-in period of the mother. Every doctor who has to attend the newborn child should be a pædiatrist to some extent if only to protect the health of the normal child.

In the present work an attempt is made to present a review of our present knowledge of the physiology and pathology of the newborn. The literature of the last decade (to the end of the year 1912) has been carefully studied for this purpose. The term "diseases of the newborn" is used in so wide a sense that not only are those diseases discussed which belong exclusively to this first period of life, but as complete a review as possible is given of all those morbid processes which may occur in this period. An attempt has been made in this matter to consider the connection with later infancy and childhood as far as possible. We realize to our sorrow that in this respect there are still many gaps in our knowledge. The diseases of the foetus, especially the malformations, are only discussed at length when they present characteristic clinical pictures. All those conditions which prevent the child surviving, and those which remain stationary during later life, are only lightly touched on. The question of nutrition, which is so extremely important during the earliest period of life, receives a fuller consideration than has hitherto been the case in similar works.

Of recent years I have been in the fortunate position of being able to collect abundant experience from the great mass of clinical material of the First University Women's Clinic in Vienna. Hofrat Schauta, at the suggestion of Escherich, permitted a newborn clinic to be established in his clinic under the care of an assistant of the children's clinic. With the conviction that the newborn child, from a human, medical and scientific standpoint, is entitled to the same skilful treatment as its lying-in mother, Schauta set up this infant clinic in the women's clinic, as he describes in his memorial to Escherich. I think that to him the specialists in children's diseases

owe the greatest obligations. I personally took charge of the newborn department at the commencement of 1911, and take this opportunity of expressing my warmest thanks to Hofrat Schauta and the gentlemen of his clinic for their great kindness to me.

Since the summer session of this year, it has also been my duty to instruct the midwives of the midwifery school in the Third Obstetrical Clinic of Vienna in the care of children. Regierungsrat Prof. Piskacek has introduced an innovation of wide significance in attaching a specialist in diseases of children for the instruction of midwives. It is hardly necessary to point out how exceedingly important it is that the midwives, who most frequently are the mothers' advisors on the ailments of children, should be well versed in the prophylactic measures in the matter of the nutrition and care of children (not only the newborn). I am greatly indebted also to Professor Piskacek and his assistants for being allowed admission to his clinic and study of the clinical material.

The illustrations in this book come in great part from the collection of the University Children's Clinic, which my honoured chief, Professor Freih. v. Pirquet, readily placed at my disposal. For a large number of the photographs I am indebted to Dr. E. Nobel. I must also express my gratitude to all those gentlemen who allowed me to reproduce their figures.

A. v. REUSS.

Vienna,
January, 1914.

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PART I

Physiology

(A) WEIGHT AND BODY MEASUREMENTS

Weight at Birth

ACCORDING to many authors the average weight after birth of the normally developed child amounts to 3,250 gm. in boys and 3,000 gm. in girls (Gundoban). Too much stress must not be laid on the averages, as great divergencies can take place in either direction without any pathological factor being involved. The normal weight at birth may be considered as varying between 2,600 and 4,300 gm. Apart from cases of premature birth, children with low weights at birth (i.e., anything under 2,800 gm.) may belong either to the class of the weakly and debilitated, or they may, in spite of their much smaller size, be considered as perfectly normal; they may develop without any set-backs, often much better than many children who have a much higher weight at birth.

The upper limit of normal weight is 4 kg. Winckel classified infants of a higher birth weight as "giant children," like those described by Fuchs, Jacoby, Schubert, Oyamada, and others. Although a birth weight of between 4 and 5 kg. is not rare, a higher figure is rarely gained. Among 30,000 births he (Winckel) found no child weighing as much as 6 kg. Oyamada's statistics, from the Munich maternity clinic, taken from 34,839 births, show a weight of

| | |
|-------------------|-----------|
| over 4,000 gm. in | 821 cases |
| " 4,500 " " | 96 " |
| " 5,000 " " | 14 " |
| " 5,500 " " | 1 case |

It is remarkable that among these infants only four were born asphyxiated, thus giving a maximum mortality of 4.08 per cent.; the chances of life, in this class of "giant children," are therefore very good. With higher birth weights (6 to 9 kg.) the prognosis is less favourable. The majority of such cases of high weight, described up to the present, were stillborn in a macerated condition. The highest weights on record are 10,733 gm. (body length 76 cm.) and 11,300 gm. (body length 70 cm.)—Duhols. The following statistics of Hecker, extending over a period of 20 years, give a good bird's-eye view of the weights of children at the beginning of life:—

| 2,500—3,000 gms. in 25·8 per cent. of all infants | | | | |
|---|---|------|---|---|
| 3,000—3,499 | " | 44·4 | " | " |
| 3,500—3,999 | " | 22·7 | " | " |
| 4,000—4,499 | " | 4·6 | " | " |
| 4,500—4,999 | " | 0·45 | " | " |
| 5,000—5,499 | " | 0·02 | " | " |

Heidemann found the following weights in 300 children :

| 1,500—1,999 gms. in 0·5 per cent. | | | | |
|-----------------------------------|---|------|---|---|
| 2,000—2,499 | " | 6·7 | " | " |
| 2,500—2,999 | " | 17·8 | " | " |
| 3,000—3,499 | " | 40·5 | " | " |
| 3,500—3,999 | " | 17·0 | " | " |
| 4,000—4,499 | " | 2·0 | " | " |

Hence it follows that birth weights of 3 to 3½ kg. are the most common.

Changes in Weight during the first weeks of Life (Physiological Loss of Weight)

During the first two to four days newly born children lose in weight. Exceptions to this rule are so unusual that observations, such as those of Laure, of children who do not lose in weight, or who regain their birth weight on the second or third day, can hardly be considered as physiological. Pies, out of 150 cases did not observe any in which this loss did not take place, and also the author, with still greater material at his disposal, failed to find a single case in which Laure's statements were supported.

Under normal conditions this loss in weight lasts two to three days so that on the third or fourth day the weight reaches its minimum. In rare cases this occurs on the second day. After the fourth day it only takes place either when there is deficiency in the supply of nourishment or of fluid, in fact, under circumstances, which can no longer be regarded as truly physiological though giving no cause for anxiety.

The usual decrease of weight varies between 150 and 300 gm. In very favourable conditions of nursing and with an early supply of nourishment and fluid this loss in weight may amount merely to 100 gm. or even less; quite frequently, however, it is considerably greater. A loss of 700 gm. is given as an outside figure (Czerny-Keller), which is a very uncommon occurrence, probably due to markedly pathological conditions. Losses of 400 to 500 gm. are nevertheless observed in children having no clinically recognizable disorders, who subsequently develop in a perfectly normal manner. The loss in weight is generally proportional to the absolute body-weight, varying from 6 to 9 per cent. of the latter. In this connection the statements of different authorities are very divergent. The loss is usually less in children of multiparae than in those of primiparae; this is probably due to the better secretion of milk usual in the former. The following tables illustrate these proportions:—

HART'S TABLE.

(a) *Children of women who have not previously nursed.*

| Weight at birth | Number of children | Average total loss in weight |
|-----------------|--------------------|------------------------------|
| 2,010—2,500 gm. | 31 | 199.4 gm. = 9.3 per cent. |
| 2,510—3,000 " | 110 | 304.58 " = 7.1 " |
| 3,510—4,000 " | 42 | 223.75 " = 6.0 " |
| 4,010—4,500 " | 8 | 228.75 " = 6.6 " |
| Average of | 391 | 225.42 " |

(b) *Children of women who have previously nursed.*

| Weight at birth | Number of children | Average total loss in weight |
|-----------------|--------------------|------------------------------|
| 2,010—2,500 gm. | 31 | 171.80 gm. = 7.6 per cent. |
| 2,510—3,000 " | 43 | 173.62 " = 6.5 " |
| 3,010—3,500 " | 75 | 182.50 " = 5.6 " |
| 3,510—4,000 " | 46 | 228.70 " = 6.1 " |
| 4,010—4,500 " | 6 | 256.00 " = 5.9 " |
| Average of | 191 | 201.20 " |

PIES'S TABLE.

| (a) FEEDING | | | (b) MILEAGE | | |
|-----------------|--------------------|--------------------------|--------------------|-------------------------|--|
| Weight at birth | Number of children | Average loss | Number of children | Average loss | |
| —2,500 gm. | 3 | 240 gm. = 11.3 per cent. | 4 | 195 gm. = 8.2 per cent. | |
| 2,510—3,000 " | 20 | 235 " = 8.5 " | 8 | 480 " = 8.3 " | |
| 3,010—3,500 " | 50 | 265 " = 9.0 " | 15 | 295 " = 8.6 " | |
| 3,510—4,000 " | 32 | 260 " = 9.7 " | 10 | 315 " = 8.7 " | |
| 4,010—4,500 " | 4 | 245 " = 8.4 " | 5 | 366 " = 8.5 " | |
| Average of | 108 | 266 " = 9.0 " | 42 | 320 " = 8.0 " | |

The loss in weight is partly dependent on the technique of the administration of nutrition, as shown by the fact that the average loss is smaller when suckling begins on the first day and is continued at frequent intervals. The observations of Sadovsky, brought forward by Gundobin, deal with children who were put to the breast twelve times during the first twenty-four hours, later two-hourly by day and four times during the night; on their average body-weight of 3,500 gm. the loss amounted to 188 gm., = 5.6 per cent. (maximum 6.8 per cent., minimum 4.7 per cent.). This is considerably less than with the infants observed by Pies who only had five meals in twenty-four hours and none on the day of birth. It must not be concluded that the latter method of feeding, as will be shown later, is not by far the better. A greater loss in weight certainly does not harm the child. It follows, moreover, from the observations of Heidemann, that four-hourly feeds give excellent results with regard to the duration and amount of the (physiological) loss in weight. He found an average loss in weight of 246.6 gm. during 3.03 days; nevertheless, almost 75 per cent. of all these infants regained their original weight in the course of seven and a half days on the average.

Landois believes the ligation and division of the umbilical cord to be a factor controlling the decrease of weight. This he finds (out of 50 infants) to be less in cases where division is performed

early than when late (5.9 to 7.4 per cent.). The greatest loss (8.8 per cent.) he finds in jaundiced children. A possible sequence of cause and effect between icterus and loss in weight is suggested on page 57.

The loss in weight depends above all on the physiological fact that a newly born child during the first few days excretes more than it assimilates. The average amount of meconium is usually given as 70 to 90 gm. (Cameron). Hirsch finds distinctly higher amounts, mostly between 150 to 200 gm. (maximum 320 gm.). He therefore thinks that the loss in weight is chiefly due to the excretion of meconium. It is, however, certain that other important factors are concerned. The child passes urine during the first few days, even before any fluid is taken, and also later in amounts which are sometimes greater than the absorption. After all, the part played by the passing of urine in diminishing weight may be relatively small. Of similarly small account is the water lost by drying of the stump of the umbilical cord. One factor in the loss of weight the importance of which must not be underestimated is the vomiting which affects many children, during the first few days, of a fluid consisting of secretions from the amnion, the organs of generation of the mother, and of blood.

It is only necessary to examine the newborn child thoroughly once during the period of losing weight to perceive that a loss in the body substances which are derived from the foetal period, is not all that takes place. The post-partum tightness or succulence of the infantile skin undergoes a fundamental change up to the day of minimum weight, while the original turgidity of the skin is lost so that it becomes flabby and wrinkled. From this it may be concluded that the body has suffered a loss in its own constituents. Researches on metabolism have shown this to be due partly to a loss in solid substances—probably chiefly fat and glycogen but also of protein; the greater part, however, is not concerned with formed tissue but is due to the loss of water through the lungs and skin (Birk). The great significance of the excretion of water in the condition of physiological loss in weight is shown by the strong, persistent and immediate inclination of the body to retain water during the first few days of suckling, and also by the fact that on a diet consisting chiefly of water a rapid gain in weight can take place. To use a somewhat forcible comparison it is as if a sponge, which has been squeezed dry, is again brought into contact with water.

If several measurements be taken in the course of one day it will be seen that the curve of the body weight falls practically in a straight line up to a point at which the intake of fluid is somewhat increased. It certainly appears to be independent of the excretion of urine and faeces. The fall in the curve is generally steepest on the first day after which it becomes less steep until the point of minimum weight is reached. The amount lost is considerably greater on the first day than on the second day.

The fluctuations in the amount of loss in weight probably

depend therefore, apart from the previously mentioned variable quantities (mucosium, &c.), chiefly upon the high water content of the body directly after birth; this, reckoned absolutely, is greater in the case of large infants as might be expected, but among children of the same weight individual cases show a considerable amount of variation. It thus appears that the loss of water may vary considerably among cases in which the water content of the tissues is approximately the same and in which the environment is similar. In this connection it is hard to differentiate between normal and pathological processes. On the whole it is certainly permissible to attribute the marked changes in the water content of the organism (as expressed in the physiological loss) to delay characteristic of the newborn child in assuming the function of maintaining equilibrium when the supply of H_2O is deficient or small. At all events it is necessary to realize clearly that we are here dealing with purely physiological processes. Of such a character is the child's small inclination for drink during the earliest days and the originally gradual increase in secretion of the mammary gland. There is absolutely no reason for treating the loss in weight therapeutically unless it be excessive in amount or continues beyond its normal time. In the latter case the incipient dryness can be hindered by the administration of indifferent liquids.

The behaviour of the body weight during the period immediately following that of minimum weight varies greatly in individual cases. The chief factor in this connection is the amount of nourishment and fluid taken by the child. The power of suction of the child, the milk secretion of the breast and especially the amount of suckling, are undoubtedly of great influence on the curve of weight. It also appears that even at this early period individual differences in the energy of growth are evident and also in the power of water retention, &c. It is also very possible that slight disturbances, which can hardly be considered pathological, have some influence (e.g., considerable intestinal irritation, &c.).

Pies divides the course of the change in weight of the newborn child into two types. The first represents the course described by Budin as the normal curve; after a fall lasting two to three days the weight increases regularly so that the original weight is regained in about ten days; the curve shows an acute angle. In the second type it is more rectangular or may show a gentle bend. In either case the child does not regain its original weight until much later (often not for some weeks). The classification of such curves as physiological is against the usual view, which considers that in the perfectly normal course of events the original weight is regained in ten days. Gundobin, moreover, teaches that the newborn child should regain its weight in between three and eleven days; the author, however, fully supports the conception of Pies. Statistics dealing with the nutrition and health of children when they have regained their original weight, are drawn exclusively from maternity institutions from which the mothers have usually been discharged between

the eighth and tenth day. It is therefore impossible to give a definite decision with regard to this period. It is an indisputable fact on the one hand that children, who fulfil the aforementioned conditions respecting their changes in weight, may subsequently exhibit disturbances; on the other hand, children who only recover their original weight after two to three weeks may not only show all the signs of good health but may develop remarkably well. This will be admitted by anyone who has seen both the figures and the children themselves.

If the original weight be regained within the first week it is due to exceptionally favourable suckling unless the weight has been raised excessively by overfeeding. Bullin's curve, which regains its initial height on the tenth day or earlier, can therefore be given as the ideal curve without further considerations, though it must not be considered as the only normal weight curve, for so long as the weight as shown by daily weighings increases constantly it shows a constant tendency to increase from the point of maximum weight the curve may be considered normal. With Pies one can also describe as normal a curve in which (1) a period of no change in weight for several days after the minimum is followed by a slow, irregular, step-like rise, or (2) the curve rises quickly for a few days, then falls and proceeds in an irregular and variable manner. A second marked loss in weight cannot be regarded as physiological. If the changes in weight be traced by means of four-hourly weighings it will be seen that the weight, which fell regularly during the earliest days, now rises in zigzags or in waves. A steep ascent is especially to be found after the first increased meals, this being sometimes followed during the night intervals by a marked fall. If a twenty-four-hourly average is taken in its entirety, a good ascending line is generally obtained, in spite of many wavering curves.

Body Proportions

The body length of the newborn child varies approximately between 47 and 54 cm. Female children are, on the average, somewhat lighter and shorter. The exact determination of the length of the body of an infant is a matter of considerable difficulty even when a measuring table is used. The measurements of Camerer lead him to the conclusion that the body length does not increase during the first few weeks of life and is sometimes even diminished. He explains this remarkable phenomenon by the following facts: The skull of the fully developed foetus is somewhat elongated during birth and its length may be further increased by the caput succedaneum. Camerer therefore considers the usual figures for the length of newborn children as being about 1 to 2 cm. too high, since the deformation of the skull has not been allowed for. The actual length of the newborn infant is in his opinion 48 to 49 cm. This mistake may easily be avoided if the measurements be taken after the caput succedaneum has disappeared and after

the skull has regained its normal conformation (that is to say, after one to two days). In opposition to the view of Cameron, Varior finds an average length of 40.5 cm. at birth and one of 51.8 cm. on the tenth day.

Growth in length during this period is hardly of any practical importance, the differences submitted being at all events so small that it is not possible to draw conclusions from them within so short a time.

The relative sizes of the different parts of the body of the newborn child and their relation to the corresponding measurements of the adult are illustrated in the following tabular statements from Weissenberg:—

| BODY MEASUREMENT | BOYS | | | GIRLS | | |
|-----------------------|------|------|---------|-------|------|---------|
| | Min. | Max. | Average | Min. | Max. | Average |
| Length of body | 471 | 540 | 508 | 452 | 520 | 500 |
| Circumference | 420 | 520 | 480 | 420 | 500 | 480 |
| Crown to shoulder | 112 | 135 | 124 | 101 | 131 | 123 |
| " tail | 112 | 135 | 124 | 100 | 134 | 123 |
| Breadth of shoulder | 90 | 111 | 102 | 86 | 120 | 104 |
| " hips | 70 | 87 | 78 | 68 | 81 | 72 |
| Circumference of head | 305 | 353 | 327 | 290 | 350 | 320 |
| " chest | 255 | 310 | 282 | 250 | 300 | 285 |
| Length of trunk | 195 | 240 | 214 | 180 | 240 | 214 |
| " arm | 195 | 235 | 214 | 181 | 225 | 210 |
| " leg | 180 | 222 | 205 | 170 | 218 | 205 |
| " hand | 58 | 70 | 64 | 58 | 75 | 64 |
| " foot | 75 | 85 | 78 | 67 | 85 | 78 |

The peculiarities of proportion characteristic of the newborn child are as follows: (1) The circumference of the body is less than the length. (2) Not only the crown to tail length, but also the trunk alone is longer than the leg. (3) The trunk length alone exceeds that of the arm. (4) The arm is longer than the leg. (5) The circumference of the head is greater than that of the chest, though sometimes the head and chest measurement are almost equal, and in the case of very powerfully built children the chest measurement may sometimes exceed that of the head.

A description of the peculiarities of proportion includes the skull, which suffers various deformities during the birth process. The true intra-uterine (pre-natal) shape of the skull is oval; in its passage through the generative canal it undergoes a change in form which is known as the "moulding" of the skull. The subsequent altered appearance is due partly to the disproportion between head and generative canal, and partly to the degree of hardness of the skull and to the intensity of the uterine contractions. Stumpf is of opinion that the amount of "moulding" is determined chiefly by the plasticity of the individual head, and that the deformity is due entirely to the soft parts of the generative canal.

The configuration is made possible by the elasticity of the skull and its overriding at the sutures. Corresponding to the frequency

of the occipital presentation this overriding takes place, especially in the parietal bones in the region of the sagittal suture. The frequently somewhat flattened parietal bone which is placed posteriorly during the birth is pushed under the anterior one. Usually the occipital and frontal bones are also pushed under the anteriorly placed parietal or under both parietals. In a condition rarer than this the two halves of the skull are pushed together in a horizontal direction, so that the one half of the forehead projects beyond the other. In these cases one parietal eminence is posterior to the other, the temporal fossae are unequal, and one parietal bone is flattened (Olshausen). The shape of the skull is also usually altered in such a way that its breadth is reduced by pressure or it is



FIG. 1.—Moderate moulding of the skull in occipito-posterior position of the head. (After Baum.)



FIG. 2.—Marked moulding of the skull in occipito-posterior position. Note the bag-like prominence of the posterior part of the skull marked off by a depression in the region of the large fontanelle. (After Baum.)

elongated in a cylindrical manner, the latter condition arising especially in cases of evenly contracted pelvis. When a large caput succedaneum develops over the foremost parietal bone the skull will show an extreme, very disfiguring deformation. These alterations of the skull may still be counted physiological since no injury is caused (figs. 1, 2 and 3). The configuration only causes a slight diminution in the total volume of the head, under which circumstances the cerebrospinal fluid flows elsewhere.

In the course of the first few days the alterations in the shape of the head disappear and have usually quite gone by the seventh



FIG. 3.—Marked moulding of the skull in original posterior position of the head.
(After Braun.)

or eighth day. When the head of an adult resembles a typical configuration it must be considered as hereditary or as the result



FIG. 4.—Moulding of the head in face presentation. (After Braun.)



FIG. 5.—Moulding of the head in face presentation. (After Braun.)

of more lasting intra- or extra-uterine influences, not as a retained configuration. The types known as dolichocephalic and brachycephalic are probably already differentiated before birth (A. Müller). It thus appears that the peculiar shapes of head found after rare presentations in so far as they persist are not the consequences, but more the causes of the presentation concerned (figs. 4 and 5). While skulls born by the occipital presentation are oval, or rather, elliptical and dolichocephalic, those after birth by the anterior frontal presentation are characterized by a high, exceptionally vertical forehead, short fronto-occipital diameter, and considerable height of the head; these skulls are chiefly brachycephalic. The frontal presentation



FIG. 6.—Shape of skull at birth in breech presentation. (After Barnes.)

gives rise to a bridge-shaped protuberance on the forehead; the shortened diameter is the mento-occipital; to balance this the fronto-occipital diameter is considerably lengthened. The skull of a child born in a breech presentation is round and brachycephalic; this condition is particularly noticeable owing to the absence of the caput succedaneum (fig. 6); in these skulls displacement of bones are least common (Olschusen).

The average diameters of the skulls of newborn children are as follows (Schäfer):—

- (1) Suboccipitobregmatic (from the posterior edge of the posterior fontanelle to the anterior angle of the anterior fontanelle): 9 cm.
- (2) Frontooccipital (from the glabella to the external occipital protuberance): 11 cm.
- (3) Mentooccipital (from the point of the chin to the farthest point of the back of the head): 13 cm.

(4) Vertical diameter (from vertex to base of skull): $12\frac{1}{2}$ cm.

(5) Bi-parietal diameter (between the two parietal eminences): 9 cm.

(6) Bi-temporal diameter (between the most distant points of the two osseous sutures): 8 cm.

Individual differences are very considerable in this connection.

Average weights of the internal organs of the newborn child in grammes (after Gundoban):—

SIZE OF THE SURFACE OF THE BODY.

| | |
|---------------------|--|
| Brain | 369 (\bar{x}) = 354.5 (\bar{x}) |
| Heart | 17.24 (\bar{x}) = 16.5 (\bar{x}) |
| Lungs | 57 (right 32, left 25) |
| Liver | 120 = 120 |
| Pancreas | 2.05 |
| Spleen | 7.2 |
| Kidneys | 11 = 12 |
| Suprarenals | 2.5 |
| Testicles | 0.8 |
| Epididymides | 0.12 |
| Ovaries | 0.2 |
| Thyroid | 1.6 (max. 2.8, min. 1.1) |
| Thymus | 1.7 |

It appears from the following tabular statement of Ssyischeff that the ratio of total body surface to weight is greatest in the case of the newborn child, this being specially marked in cases of premature birth. In the course of growth this ratio steadily diminishes. The appreciation of this fact is important, since it enables us to understand the high energy requirements of the young infant which are dependent on the relative amount of the heat loss.

| Age | Weight gm. | Amount of body surface in sq. cm. | Body surface per kg. body weight in sq. cm. |
|---------------------------------|---------------|--------------------------------------|---|
| Four days old, prematurely born | 1,105 | 1,266.4 | 841.4 |
| Newborn | 2,000 | 1,425.0 | 704.0 |
| Fifteen days old | 2,680 | 2,120.0 | 711.0 |
| Six months old | 5,112 | 2,701.0 | 526.2 |
| One year old | 9,005 | 4,800.0 | 527.0 |

(B) METABOLISM AND DIGESTION

The Chemical Composition of the Newborn Child

The body of the newly born child cannot differ essentially in gross chemical composition from that of the infant some weeks or months old (Camerer). Camerer and Söldner examined the bodies of six newborn children, and maintained from the agreement of their separate analyses that the following are the average values for an infant:—

| | Body weight | Water | Solids | Fat | Ash | Protein | Extraneous |
|--------------------------------------|-------------|-------|--------|------|-----|---------|------------|
| Absolute values | 1,820 | 2,020 | 705 | 528 | 75 | 350 | 42 |
| 100 gm. body substance contain | — | 21.6 | 8.2 | 12.1 | 2.7 | 11.7 | 1.5 |
| 100 gm. dried body substance contain | — | — | — | 41.8 | 9.4 | 49.4 | 5.5 |

Elementary Composition.

| | C | H | N | O |
|--------------------------------------|-------|-------|------|--------|
| Absolute value | 429.6 | 67.15 | 33.8 | 147.45 |
| 100 gm. body substance contain | 15.9 | 2.45 | 1.98 | 5.29 |
| 100 gm. dried body substance contain | 59.5 | 8.4 | 7.0 | 18.7 |

The body of the newborn child is relatively rich in water and fat, and correspondingly poor in nitrogenous substances and in ash. The relative large water content is a residue from the foetal period.

According to Fehling, the younger the foetus the richer it is in water. The deposition of solid constituents causes the percentage water content of the body to fall between the sixth week and the ninth month from 67.54 per cent. to 74.7 per cent. The dehydration expressed in these percentages continues into extra-uterine life, so that the adult body has a water content of about 58.5 per cent. The proportion of ash shows a corresponding increase.

The composition of the ash is generally constant from the fourth month of foetal life onwards ("the constant of the relative ash content of the foetus"). Only calcium is deposited in much larger amounts (32 to 34 per cent. of the total ash in the fourth month, 40 per cent. in the full-time foetus); the foetus, therefore, requires a large amount of calcareous matter. According to Hugonienong, a particularly large amount of iron is also taken up during the last third of intra-uterine life. He therefore recommends that the mother should be provided with a diet rich in calcium and iron during the later months of pregnancy.

The following table from Birk shows the percentage composition of the inorganic constituents of the body:—

| | |
|--------------------------------|--------------------------------|
| CO_2 = 1.16 | CaO = 40.38 |
| Cl = 4.54 | K_2O = 7.50 |
| P_2O_5 = 99.26 | Na_2O = 5.96 |
| SO_2 = 1.25 | Fe_2O_3 = 0.40 |

Very thorough investigations have been made concerning the composition of the fat, which, in the newborn child, forms half of the dry weight of the body, and about 12 per cent. of the total weight. These discoveries are of interest, since certain pathological conditions of the subcutaneous tissue, which occur during the first weeks of life, are probably connected with the quality of the fat. Fat consists essentially of triglycerides of palmitic and stearic acids (solid fatty acids) and of oleic acid (liquid fatty acid); in comparison

with these the part played by the volatile fatty acids is quantitatively small. The distribution of the fatty acids in the child is different from that in the adult. Langer found

| | In the adult | In the child |
|---------------|----------------|-----------------|
| Oleic acid | 56.8 per cent. | 60.75 per cent. |
| Palmitic acid | 3.16 " | 23.97 " |
| Stearic acid | 2.94 " | 1.18 " |

These researches, which show the relatively small proportion of oleic acid in the fat of the infant, were extended by Knöpfelmacher, who made similar researches during infancy. He found

| In the newborn | 44.40 per cent. liquid fatty acids |
|---------------------------|------------------------------------|
| " the 10 months old child | 61.99 " |
| " the 12 months old child | 59.22 " |

The proportion of oleic acid increases with the increasing age of the infant, as is shown by an increase of the originally low iodine value (Knöpfelmacher and Lehndorf, Siegert, Thiemich, Jackle, Engel, Debatonkin). It amounts to about 43 to 45 in the newborn child, and to 60 to 70 in the adult.

The chemical composition of the fat involves the fact that the fat of the newborn child has a relatively high melting point (38°); this height falls with the increasing age of the infant. It is further worthy of note that the composition of the fat of the newborn child differs in different parts of the body, e.g., in the sole of the foot it contains 67.28 per cent. of oleic acid, thus approaching the composition of adult fat.

A further characteristic of the fat of the newborn child is its relatively high proportion of volatile fatty acids. According to Engel and Bode, this amounts approximately to 3 to 5 per cent. as compared with $\frac{1}{2}$ to 2 per cent. in the older infant; in the adult considerably smaller values are sometimes found. The above qualities cause the fat of the newly born child to assume the character of milk fat. Engel and Bode, who called attention to this condition, attribute it to the activity of a body (of the character of a hormone) supplied by the mother, which causes a characteristic change in the material supplied to the fetus for the laying down of fat, just as it does in the case of milk. After birth this influence continues, so that now normal human fat can develop without the perceptible cooperation of alimentary influences. The fat of the infant loses its character of milk fat at some unknown period, not later than the end of the first year.

The Process of Metabolism during the Newborn Period

Our knowledge of the process of metabolism in the organism of the newborn child is founded essentially on the results of urine analyses, which show several peculiarities, especially in respect of substances containing nitrogen. Up to now only a few researches

on metabolism are available in which anabolism has been considered as well as katabolism. The technical difficulties of such researches are particularly great during the first few weeks of life.

If we disregard for the moment the difficulties of obtaining a quantitative determination of the excretions, we nevertheless find certain difficulties in obtaining the necessary materials (e.g., colostrum) for the analytical determination of the intake.

The constituents of the undiluted milk of a woman who has been suckling for some time are no longer present in the correct physiological proportions. For this reason Birk proceeded in the following manner: he allowed the child to drink from one breast, and pumped the other breast meanwhile as dry as possible and analysed the secretion.

NITROGEN METABOLISM.

Ogler is of opinion that infants in the first fourteen days of life retain the maximum amount of N_2 , the highest value in utilization of that substance for anabolic purposes. According to his calculations the newborn child anabolizes 78.3 per cent. of N_2 administered, as compared with 40.8 per cent. in the second and third months and 23.7 per cent. in the fifth month.

Langstein and Niemann, who made the first accurate determinations of metabolism of newborn children, arrived at a different conclusion. They found a negative N_2 balance in three normal children during the first days of life. The nourishment consisted of human milk drawn off with a breast pump, beginning on the second day of life. The negative nitrogen balance lasted five, three, and two days in the respective cases; in the last, however, a negative value was again obtained three days later, and continued until the eighth day, whereas the balance in the first two cases remained positive all the time.

In a similar research Birk found a negative balance throughout the four days of observation. On the contrary, however, in the case of two children who were fed from the breast with the inclusion of the colostrum, the N_2 balance remained on the whole positive from that day forward on which the child first received nourishment (i.e., on the second or third day). Although in one case the secretion of milk was quite insufficient, the child sucked badly and lost in weight continuously. Birk also found a positive N_2 balance in children fed with cow's milk (half milk).

Although it appears that in the physiological nourishment with colostrum, the balance may continue positive, many researches accord in demonstrating that during the first days of life the excretion of N_2 , reckoned absolutely, is very high (Vogt, Langstein and Niemann, Weigert and Seelitz, Simon, Birk, &c.). The following table from Gandolin-Kotschurowski illustrates the average nitrogen excretion during the first week:—

| | | | | | |
|---------|--------|------|------|------|-------|
| 1st day | 131.12 | 166. | 7.85 | 167. | 1094. |
| 2nd " | 118.97 | " | 6.85 | " | " |
| 3rd " | 222.87 | " | 3.90 | " | " |
| 4th " | 304.44 | " | 1.90 | " | " |
| 5th " | 425.90 | " | 2.68 | " | " |
| 6th " | 425.52 | " | 1.72 | " | " |

The absolute amount of nitrogen, therefore, increases from day to day, while the nitrogen content of the urine, calculated in percentages, falls correspondingly to the increase in the amount of urine.

Birk measured total N_2 in urine during the first six to seven days of life in a number of children fed with colostrum milk, giving as the daily output 0.1471 to 0.3972 gm. "If these figures be compared with the N_2 content of urine of older infants as estimated in the literature, it will be concluded that in the newborn child the N_2 excretion is very high; in the course of the first week it sinks to about half its initial value; it then rises a second time, parallel with the N_2 used for nutrition. About the fortieth day the N_2 in the urine again reaches the height it had been immediately after birth." If the daily N_2 changes be examined more closely, it will appear that the amount in the urine on the first day is usually considerably lower than on the following days. The value increases until the middle of the first week, when a gradual fall takes place. Hence in one of Birk's cases the N_2 output rose during the first three days from 76, over 336 to 392, and sank on the fifth day to 137 mg.; in a case of Langstein-Niemann it amounted on the first day to 69 mg., on the eighth day it rose to 331, and on the ninth day to 135 mg.

In order to throw more light on the origin of the high N_2 output, we must take account of the various forms in which N_2 appears in the urine. A high value was formerly given to the urea-content of the urine of the newborn child, but it appears that the amount of urea is not so very great, since the older observers only considered the absolute values without investigating their relation to the total output. According to Hofmeyer the formation of urea increases rapidly during the first few days, so that on the fourth day the urea excreted in the urine amounts to more than four times its initial value, and approximately twice as much as on the eighth or ninth day. Schiff and Reusing, however, taking their evidence from nearly fifty cases, conclude that these proportions are by no means the rule. If the absolute values of the excreted urea be taken in the course of twenty-four hours the following figures result:—

| | p.p. | 1st | 2nd | 3rd | 4th | 5th | 6th | 7th | 8th | 9th | 10th |
|--------------------|------|-----|-----|-----|-----|-----|-------|-------|-----|-----|------|
| Reusing | ... | 50 | 60 | 160 | 520 | 900 | 760 | 790 | 610 | | |
| Schiff | ... | ... | 115 | 420 | 455 | 590 | 595 | 635 | 664 | 711 | 517 |
| Katschowsky (max.) | ... | ... | 817 | 712 | 785 | 763 | 1,414 | 1,160 | | | |
| " (min.) | ... | ... | 42 | 179 | 233 | 261 | 360 | 412 | | | |
| " average | ... | ... | 187 | 338 | 463 | 467 | 711 | 720 | | | |

According to Kentschawski, the relation of urea N_2 to total N_2 of urine is shown in the following table:—

| N | 1st day | 2nd | 3rd | 4th | 5th | 6th |
|--|------------|------------|------------|------------|------------|------------|
| Total N | 131.67 | 113.97 | 111.87 | 99.47 | 221.60 | 423.52 |
| N of the urea | 87.44 | 157.70 | 206.55 | 232.15 | 133.60 | 345.37 |
| Proportion of total N to N of the urea | 1 0.736 | 1 0.736 | 1 0.793 | 1 0.262 | 1 0.779 | 1 0.812 |

The urea coefficient, therefore, arises from 72 to 81 per cent. during the first week. In this connection there is no essential difference between the newborn child and the older infant. According to L. F. Meyer and Riesshol, the urea coefficient of these latter amounts to 60 to 80 per cent. Should this lower value be too low, as Vogt believes, then values of about 75 per cent. can also be considered normal in older infants.

While the excretion of urea rises, that of uric acid, according to Reusing, remains practically stationary apart from the third day. In any case it shows no marked progressive increase; Reusing believes that the apparently unexplained increase in excretion of uric acid which he found on the third day may be attributed to the fact that part of the uric acid already analyzed cannot be excreted immediately, owing to the shortage of fluid; on the third day, however, its excretion does occur. The average absolute value of uric acid excreted in six cases is as follows:—

| Days | 1. | 2. | 3. | 4. | 5. | 6. | 7. |
|------|------|------|------|------|------|------|------|
| | 41.0 | 41.4 | 83.0 | 39.5 | 36.6 | 46.3 | 37.3 |

According to Sjögren, the excretion of uric acid is especially high before and especially during the so-called "infant period," which is to say, according to the researches of this writer, in the urine of the second half of the first and the whole of the second day; after the infant period it also remains at a higher level than in the adult during the remainder of the first week. The ratio of uric acid to urea is:—

| | |
|-------------------|--------|
| Before the infant | 1:0.77 |
| During the infant | 1:6.42 |
| After the infant | 1:17.1 |

Reusing, however, gives the following figures:—

| | Uric acid | Urea |
|-------------|-----------|------|
| 1st day = 1 | 1.5 | |
| 2nd " | 1 | 3.2 |
| 3rd " | 1 | 5.5 |
| 4th " | 1 | 11.3 |
| 5th " | 1 | 15.0 |
| 6th " | 1 | 17.5 |
| 7th " | 1 | 21.5 |

The results of the recent researches of O. M. Schloss and Crawford are in accordance with these older figures. They found that the excretion of uric acid in newborn children was absolutely

and relatively fairly high, especially during the first three days of life, after which time the amount diminishes. Niemann gives the following results:—

| Day | 1. | 2. | 3. | 4. | 5. | 6. | 7. | 8. | 9. | 10. |
|-------|--------|-------|-------|-------|-------|-------|-------|--------|-------|-----------|
| Ur. | Traces | 20.11 | 29.07 | 99.07 | 57.53 | 44.99 | 12.23 | 29.20 | 40.50 | 50.20 mg. |
| Para. | — | 0.09 | 0.15 | 0.08 | 0.04 | 0.04 | 0.03 | 0.03 | 0.04 | 0.03 % |
| Bases | Traces | 1.73 | 52.02 | 0.81 | 2.50 | 5.81 | 4.50 | Traces | | |

In this case the excretion of uric acid was highest on the third and fourth days and then fell gradually to lower values; simultaneously the urine of the third and fourth day shows a very high concentration of uric acid (9.1 per cent.). From the fifth day onwards the excretion of uric acid assumes an approximately constant level of 30 to 40 mg. a day, although variations are noticeable at a later period.

According to Gandolin the uric acid content undergoes considerable variations during the first four days of life; considered as percentages, however, it diminishes constantly in the first six days. "The urine of the newborn child contains, both absolutely and also per cent., two to three times as much uric acid as that of children later in infancy." Kotscharowski gives the following table to show relationship of uric acid to total N_2 and to urea:—

| Days | PERCENTAGES | | |
|------|------------------------------------|------------------------------------|----------------------|
| | Of total N_2 to N of uric acid | Of N of urea to N of uric acid | Of uric acid to urea |
| 1 | 1 : 0.060 | 1 : 0.090 | 1 : 2.70 |
| 2 | 1 : 0.059 | 1 : 0.082 | 1 : 2.6 |
| 3 | 1 : 0.043 | 1 : 0.055 | 1 : 2.2 |
| 4 | 1 : 0.041 | 1 : 0.055 | 1 : 2.0 |
| 5 | 1 : 0.036 | 1 : 0.038 | 1 : 1.8 |
| 6 | 1 : 0.020 | 1 : 0.027 | 1 : 1.5 |
| 7 | 1 : 0.018 | 1 : 0.024 | 1 : 0.7 |

Birk, who determined the uric acid content of the urine in a larger number of children of the first week, in order to ascertain the influence of nutrition and the termination of the ligature and division of the umbilical cord on them gives the following figures (mg.):—

| | Days | | | | | | | | |
|----|-------|------|-------|------|------|------|------|------|------|
| | 1st | 2nd | 3rd | 4th | 5th | 6th | 7th | 8th | 9th |
| 1 | — | — | 17.37 | 0.4 | 11.0 | 10.1 | — | — | — |
| 2 | 15.5 | 46.0 | 20.5 | 18.1 | 15.4 | — | — | — | — |
| 3 | 7.7 | — | — | 5.2 | 7.7 | — | — | 11.0 | — |
| 4 | 10.5 | 28.7 | — | 47.7 | 21.4 | 21.4 | 18.4 | — | — |
| 5 | 25.25 | 15.7 | 64.0 | 14.8 | 14.0 | — | — | — | — |
| 6 | — | — | 12.0 | — | — | — | 25.4 | 14.9 | 55.6 |
| 7 | 6.0 | 8.0 | 39.2 | 36.1 | 36.7 | 37.4 | — | — | — |
| 8 | 106.0 | 23.4 | 25.8 | 20.0 | 55.4 | 21.4 | — | — | — |
| 9 | 24.0 | 20.7 | 45.5 | 27.8 | 11.5 | — | — | — | — |
| 10 | 22.0 | — | 35.0 | 40.0 | 23.7 | 20.1 | — | — | — |

If this table be examined it will be noticed that the amounts vary within wide limits, without these variations exhibiting any

regularity. According to Birk's estimations, the average amounts given by him of the uric acid excreted during the first week vary considerably.

Nor can any regularity be deduced from the researches of Simon, who determined the relation of uric acid N_1 to total N_2 . The amounts given by him for the first nine days vary between 1 and 3.3; only in one case was the relatively high value of 9.9 found on the second day, and by the sixth day this had sunk to 4.5. An investigation of Niemann (see table, p. 17) dealing with the excretion of purin bases in the urine of the newborn child, showed a daily figure of 2 to 7 mg., a particularly high value occurring on the third and fourth days, coincident with the high excretion of uric acid. Birk, on the contrary, in daily determinations, was unable to show the presence of purin bases in a single case; he did not obtain a positive result until he examined larger amounts collected during five to seven days' excretion of urine. The average values determined by him from nine cases varied between 0.19 and 0.76 mg. From these he concludes that the daily output of purin bases is so small as to be accounted a mere trace.

Sjöqvist found the excretion of NH_3 in the urine of the newborn child comparatively high, in accordance with which determinations are some made by Keller on five healthy children of one to ten days old; the amount of ammonia came to 9.5 to 12.5 per cent. of total N_2 . According to Simon the excretion of NH_3 is fairly low on the first day. It soon rises however, and reaches a fairly high value by the end of the first week. The author arrived at similar results.

The amino-acid N_3 shows considerably high values as compared with those of the adult. Simon found 5 to 8 per cent. during the first three days of life, the amounts rising in his cases to 10 to 12 per cent. on the seventh and eighth day. The author obtained the following results in the analysis of urine during the first nine days:—

| | NH_3 | Amino-acids N_3 |
|-----------------|--------|-------------------|
| 1st day | 6.4 | ... 7.1 |
| 2nd " | 7.8 | ... 8.5 |
| 3rd " | 8.3 | ... 11.5 |
| 4th " | 10.6 | ... 7.8 |
| 5th " | 12.6 | ... 4.5 |
| 6th " | 10.2 | ... 3.5 |
| 7th " | 11.8 | ... 3.4 |
| 8th and 9th day | 9.3 | ... 2.8 |

The high values of amino-acid N_3 here fall on the first four days. In the second half of the first week they approach those values which Hadlich and Grosser found in healthy older breast-fed infants.

The so-called "Residual N_2 " shows different values according to the method used in the preparation of urea. Vogt found comparatively high values, not only with newly born children but with children nourished on human milk in general. Simon also found

high values for the residual N_2 , but in his researches these fall considerably in the course of the first week, parallel with the total N_2 until the seventh day. He found, moreover, that in spite of the high excretion of amino-acids, the greater part of the residual N_2 during the first week remained undetected. Part of this will be due to the hydroxyacids of the proteins, which Simon was able to determine up to 10 per cent. and more (of total N_2) in all his cases of breast-fed children, both the newly born and healthy older infants. Since the adult only excretes 3 to 5 per cent. total N_2 as hydroxyacids of proteins, it follows that the infant reaches a value nearly twice as high. This, however, is characteristic of the infant in general, and not of the newborn child alone. Simon, on the other hand, was able to ascertain that the newly born child excreted unhydrolysed polypeptides in large amounts during the first days of life. He found in two cases:—

| | | | |
|---------------------|----------------|-----------------|---|
| In the urine of the | 1st — 3rd days | 20.18 per cent. | |
| „ | 4th and 5th | 10.3; 12.8 | „ |
| „ | 6th and 7th | 14.9; 17.0 | „ |
| „ | 8th | 13.6; 13.9 | „ |

Other nitrogenous constituents of the urine of the newborn child play a subordinate part, at any rate quantitatively. Allantoin is absent, or only appears in minute traces (Schittenhelm and Wiener, Simon). Older positive results (Proust) must have been due to unreliable methods. Amberg and Morill found in twenty-four samples from infants aged 7 to 14 days, amounts of creatinin equal to 2.56 to 3.6 per cent. of the total N_2 . Glycocoll (amino-acetic acid, glycine) was detected every day by the author in the "milk-urine" of the first week.

METABOLISM OF SALTS.

Our knowledge of the mineral exchanges during the first days of life are still very insufficient. Only three researches by Birk are available wherein children were fed with colostrum milk, ordinary human milk, and cow's milk respectively.

In the first (colostrum) research, a distinct tendency toward retention was noticed. Throughout the period of physiological loss in weight, the balance remained positive. The following percentages of the inorganic substances administered were retained:—

| | | | |
|----------------------------|-----|-----|------------------|
| Total inorganic substances | ... | ... | = 10.4 per cent. |
| Calcium | ... | ... | = 22.5 " |
| Magnesium | ... | ... | = 20.1 " |
| Potassium | ... | ... | = 62.0 " |
| Sodium | ... | ... | = 45.4 " |
| Phosphorus | ... | ... | = 20.5 " |

In this connection it is noteworthy that the highest retention occurs in the case of P_2O_5 and Ca which are substances used in bone formation, but there is also a considerable retention of alkalis.

In the case of the artificially fed child, a positive balance was also maintained and the retention was only slightly less than in the colostrum fed child. The least favourable condition of nourishment was found on the administration of ordinary human milk, where the balance was found on the negative side; this, however, was entirely due to the deficiency of the secretion. In this connection it must be emphasized that we cannot conclude from these theoretical though interesting researches that the artificial feeding of infants is preferable to the administration of ordinary human (wet-nurse) milk.

Special consideration is due to the conditions of the excretion of phosphorus on account of the relations existing between P and N, metabolism—especially uric acid metabolism. In the first excretions or urine, the phosphorus content is low. In an analysis W. Heubner found 0.0018 P ($= 0.004 \text{ P}_2\text{O}_5$); the phosphorus content of the urine was practically the same as that of the amniotic fluid, from which we may conclude that the phosphorus excreted on the first day is not due to the disintegration of the phosphorus-containing tissues, but corresponds to the normal physiological content of the body-fluid in soluble phosphates. Langstein and Niemann, who investigated the phosphorus content of the urine in the following days of life were able to ascertain that the phosphorus value generally rises from the second day onwards; after the seventh to ninth day it begins to fall again, but the low value of 10 to 20 mg. which Moll gives for the healthy infant was not reached even by the twelfth day. These observations correspond with those of Michels, who found higher phosphorus values of 22 to 29 mg. in children between the fifth and eleventh day of life. The highest values which Langstein and Niemann found in healthy children during the first week of life amounted to 105 to 138 mg. O. M. Schloss and Crawford also found a high phosphorus excretion during the first three days, followed by a marked diminution. Only Kotscharowski found a daily increase in the average excretion of P_2O_5 in twenty-four hours.

No researches have been made on the Cl metabolism during the first days of life in which the balance was taken into account. From the Cl content of the urine alone, few conclusions can be drawn. Gundobin has produced Cl analyses of Schiff, Kotscharowski, and Gein, in which the amount of Cl excreted in the first three or four days differed very little, although the amount of Cl administered during the same period had increased considerably. A distinct analogy may here be drawn with the case of the starving adult.

Kotscharowski also made estimates of H_2SO_4 , finding that the daily excretion rose during the first six days from 7.14 to 24.75 mg. The older infant does not show essentially higher amounts.

METABOLISM OF RESPIRATION.

Birk and Edelstein used a Voit-Pettenkoffer apparatus to study the respiration of a newborn child during the whole period of the physiological loss of weight; the child was artificially fed with

half milk. The factors concerned are shown in the following table:—

| Test days | Food supply | Respiratory loss | | Excretion of | | Total | Weight |
|--------------------|-------------|------------------|------------------|-----------------|------|-------|--------|
| | | CO ₂ | H ₂ O | Mecconium Urine | | | |
| First 12 hours ... | — | 28.74 | 44.18 | 5.66 | 14.0 | 22.96 | — 126 |
| 2nd day ... | 17.18 | 44.25 | 89.57 | 5.24 | 15.4 | 16.35 | — 70 |
| 3rd day ... | 220.99 | 45.12 | 153.50 | 99.83 | 14.3 | | + 30 |

The authors calculate from the results of their experiments that about 0.22 gm. C must be derived from protein, 7.87 gm. C from fat or carbohydrate, and that during the period of observation about 0.41 gm. protein and 10.2 gm. of fat must have been burnt. The greater part of the substance lost from the body consists of the water excreted by the skin and lungs.

Cramer estimates a loss of about 20 gm. of C during the first three days of life, for which result 25 gm. of body fat must have been broken down. This calculation depends upon Cramer senior's work on a healthy breast-fed infant. The results of his observation are as follows:—

| Day | Intake of milk | Output | | Respiratory output | Total output | Weight |
|-----|----------------|--------|--------|--------------------|--------------|--------|
| | | Urine | Feces | | | |
| 1 | 10 gm. | 48 gm. | 54 gm. | 68 gm. | 162 gm. | — 187 |
| 2 | 91 " | 53 " | 26 " | 29 " | 158 " | — 62 |
| 3 | 247 " | 122 " | 8 " | 85 " | 360 " | — 23 |
| 4 | 337 " | 220 " | 5 " | 92 " | 325 " | + 25 |

Essentially lower figures for the "perspiratio insensibilis" are given by Cramer, who is of opinion that "the gaseous excretion in the newborn child is considerably less, both relatively and absolutely, than in the older infant, owing to the decreased intensity of its metabolism and of its reflexes, and also on account of the shortage of water."

Scherer is of opinion that the intensity of the respiratory gaseous exchanges in the newborn is considerably higher than in the adult.

| | | | |
|------------------|-----------------|-----|---------------------------|
| In the child ... | CO ₂ | 350 | $\frac{350}{470} = 0.702$ |
| | O ₂ | 470 | |
| In the adult ... | CO ₂ | 216 | $\frac{216}{243} = 0.89$ |
| | O ₂ | 243 | |

From this he draws the following conclusions: the relatively low respiratory quotient of the newborn child is connected with its great consumption of O₂. With a lower surrounding temperature the gaseous exchanges are more rapid and the O₂ consumption is considerably raised, so that the respiratory quotient sinks, on the average, to 0.582. The gaseous exchanges fall a little in the first hours after birth; from the ninth hour until the first half of the second day it begins to rise quickly, from which time a considerable but more gradual rise in the gaseous exchanges takes place.

Babak states that the rise of gaseous exchanges on the first day after birth is "a sign of great activity on the part of the chemical heat regulation which ceases altogether or regains sooner or later its average condition, according to the time after birth when the physical regulation of heat develops its activity."

If an attempt be made to deduce from the above experiments a general picture of the characteristic features of the metabolic processes of the newborn child, the result arrived at will be somewhat as follows: During the first days of life the organism gives off considerable quantities of water, which, reckoned absolutely, may be greater or less than in the older infant—owing to the smaller intake of water this cannot be at once replaced. Coincident with this there occurs a disintegration of the body substances; here the N_2 -free substances (especially the fats) are, quantitatively, those most affected. These processes are to be considered solely as a result of insufficient intake of food. On this account they cannot, of course, be looked upon as pathological, as reserves are present to provide the organism with sufficient material for combustion during the first days. The conditions affecting the nitrogenous substances are less clear; the figures showing the total N_2 and urea excreted, are remarkably high during the first days. Czerny and Keller explain these facts also as due to insufficiency of food alone. This involves the fact that the body is compelled to burn material derived from its own substance. "Since fat is contained in the body in large amounts this constituent is burnt in greatest proportion, thus, in the total metabolism, the protein is spared as far as possible. On this account the total N_2 excretion in the first days is lower than when food is available in amounts adequate to supply the needs of the body. This also involves the retention of the end products of metabolism, probably on account of the insufficient intake and output of water during the first days. After this time (which may be considered as a period of hunger or at any rate of insufficient nutrition), a gradual increase in the excretion of N_2 occurs in the newborn child, just as it would in the adult after a period of starvation. This lasts until the body has adapted itself to the large size of the intake of N_4 . The characteristic features of metabolism given above are most pronounced in children who have been nursed by their own mothers, though they are also present in the case of children fed with wet-nurse milk or cow's milk."

The unusual proportions in which the nitrogenous substances are present in the urine and relatively high value of the residual N_2 are not in contradiction with the suggestion that the disintegration of body protein is only a consequence of starvation conditions. Experiments on metabolism have shown that the N_2 balance in breast-fed infants must remain by no means negative after the second day. Thus it is hardly justifiable to describe the period of high N_2 excretion as a time of protein hunger; nevertheless we find an unusually high excretion, not only of N_2 , but also of the products of incomplete protein hydrolysis. This appears to indicate that,

during the first days of life, a peculiar kind of tissue disintegration takes place, which is to a certain extent independent of the intake of food. The high excretion of phosphorus, also, during the first week probably indicates a previous disintegration of body substance, which differs essentially from the changes occurring in the course of inanition (Langstein and Niemann). The incompleteness of the protein hydrolysis is due, according to Simon, to the relative poverty of the body in ferments, in consequence of which the body is unable to deal with the products of protein decomposition which overwhelm its metabolism. It is possible that a relative insufficiency of function on the part of the liver may also be of importance in this connection, and that toxic agencies which have passed from mother to child at the time of delivery (toxins of pregnancy) may take part in causing the decomposition of tissue. We find ourselves still in the realms of hypothesis concerning the nature and meaning of the period of loss of body-substance at the beginning of extra-uterine existence. It is possible, however, that many ætiologically obscure pathological conditions of the first days of life may lie in these hitherto unexplained processes of metabolism.

A peculiar significance is attached to the metabolism of uric acid and purin substances. As the majority of the aforementioned analyses would indicate, there is a comparatively large excretion of uric acid in the course of the first week of life. If the results of the experiments do not agree in all points, this is probably due to the fact that the excretion takes place irregularly. It is more than likely that the formation of uric acid is relatively large in the newborn child. Since the colostrum is fairly rich in cells, it might be thought that the uric acid was derived exogenously from the nuclei of the food. According to Birk's comparisons between infants fed on colostrum, human, and cow milk respectively, it would appear that the form of diet has little or no effect upon the amount of uric acid excreted by the infant. It must therefore be an instance of endogenous uric acid. The parallel between the excretion of uric acid and of phosphorus, observed by various authors during the first days of life, points to a common origin from cell nuclei. Brugsch and Schittenhelm, who observed a marked increase in the uric acid in the urine of the newborn child, presumed a connection between this excretion and the destruction of leucocytes, which had been taken by Horbaczewski to be the explanation of the uric acid infarct. In the destruction of cells, the nuclear substances are set free; the nucleoproteins of these substances yield purin bases, which form the mother substance of uric acid. These flood the organism suddenly, giving rise to a considerable increase in the formation of uric acid, and, consequently, to its increased excretion in the urine. Niemann explains the temporary increase in the excretion of purine bases as being due to an incomplete disintegration of purin bases owing to the rapid change of the nuclei of the leucocytes. Birk attempted to throw light on the influence of leucocytes, as Czerny and Keller had suggested, by

comparing the excretion of uric acid in cases of early and late umbilical ligation. Corresponding to the larger amount of blood which the child receives from the placenta in the latter case, the number of leucocytes inundating the body is increased, from which it follows that the amount of uric acid excreted should be greater; Birk's researches, however, gave no definite result.

The increased excretion of uric acid is mainly connected with the so-called "uric acid infarct." This term is used for the formation of a red precipitate in the region of the pyramids of the kidney. According to Flensburg it consists of ammonium triurate; according to Gundobin-Ssumzoff chiefly of uric acid, and to a lesser extent of sodium urate and calcium oxalate; altogether the infarct contains 96.3 per cent. organic matter. Czerny and Keller discussed the genesis of uric acid infarct in a paper based on an exhaustive study of the literature. They correlate the facts ascertained up to about the year 1904 in the following words: "A greater hyperleucocytosis takes place in the first days of life in infants whose umbilical cords have been ligatured late than in those ligatured at an earlier period. The uric acid content of the acid urine is much greater during the infarct period than at any other time in later life. A somewhat hyaline substance is present in the urinary tubules as a product of the cellular secretion of the convoluted tubules; this behaves like threads in a concentrated salt solution and the salts of uric acid collect on it. This last phase in the formation of infarct, so far as the process concerns the kidney, is made apparent by the formation of organic elements more or less thickly covered with urates. If we were to find the connection between the increased excretion of uric acid and the persisting hyperleucocytosis—irrespective of whether the uric acid arises from the disintegration of leucocytes or as a metabolite of the same or whether it is in any other way connected with the formation of new leucocytes—it would then merely explain to us why material for the formation of uric acid is present in such quantity during the first days of life. It is not clear, however, why there should be an increasing excretion of uric acid and a typical uric acid infarct which never occurs in the adult, although, under pathological conditions, the same factors might be present which might be connected with the formation of infarct. These are hyperleucocytosis, increased excretion of uric acid, acid reaction of the urine, and abundant albuminous secretion by the kidney."

It is true that Spiegelberg was able to show that the adult dog was able to break down uric acid to a greater extent than the young animal. When sodium urate was injected into the blood of the newborn puppy the formation of infarct resulted; in the adult dog this condition could not be produced. In man, however, this function of splitting up uric acid seems to be already fully developed in the first days of life (Schittenhelm). It is possible that it is a question of the insufficient solvent power of the infantile urine for uric acid. The researches of Spiegelberg appear to point to such a condition.

The concentration of the urine is certainly sufficient to explain the precipitate of urate in the urine, but it does not account for the formation of a precipitate in the kidney, for a kidney infarct never occurs in older persons under similar conditions.¹

The connection between the formation of infarcts and the excretion of uric acid cannot yet be considered as fully explained. It is, however, permissible to state, on the basis of the aforementioned researches, that the two processes are probably connected with one another, and are both due to a disintegration of cellular elements. The proportion of uric acid excreted always varies in individual cases according to the washing out of the precipitated urate.

The opinion put forward by Virchow that the infarct in the kidney only occurred in infants who survived delivery has been disproved, since these have also been observed in the kidneys of stillborn children, although less frequently. In the post-mortem examination the uric acid infarct is found most frequently in children between two and five days old; the excretion can, however, last several weeks (Miller).

The excretion of uric acid in the urine is not only chemically recognizable, but is also shown by the passage of an opaque urine which is often seen to contain a red granular sediment immediately after leaving the bladder. We therefore speak of a kidney infarct and a urine infarct; by the latter are indicated the masses of urate passed in the urine which are frequently visible as a brick-red powder, and which appear under the microscope partly as an amorphous and partly as a crystalline precipitate. The phenomenon of the so-called "infarct urine" must be looked upon always as a physiological process, even though it may be absent in some cases. Gundobin looks on the kidney infarct as a pathological occurrence, but it is very doubtful if he is correct in this assumption. Certainly the infarct is only found in a certain percentage of cases (20 to 42 per cent. of those born living), but this may be explained by the fact that a large number of the infants experimented on reached the post-mortem room before the formation of the infarct.

THE URINE OF THE NEWBORN CHILD.

The amount, concentration and appearance of the urine during the first days of life are dependent on the variable intake of fluid at the time. Other important items are the absolute body weight, the absolute and relative water content of the body, and the water losses through the kidney which are independent of the intake of fluid.

¹ SUPPLEMENT DURING CORRECTION OF PROOFS.—This assertion is not strictly accurate. Uric acid infarcts have also been found in the kidneys of older persons, especially in consequence of leukaemia. (M. B. Schmidt, "Ueber Harnsäureinfarcte nach Zellerfall," *Zeitschrift f. allg. Pathol. u. path. Anat.*, 24, 1913, 407.) These arise owing to the disintegration of leucocytes in the circulation; the increased breaking down of substances rich in purine leads to an increase of uric acid both in the blood and urine.

The following table from Reusing shows the amount of urine passed during each twenty-four hour period:—

| After birth — | 1 c.c.m. | 54 c.c.m. |
|---------------|----------|-----------|
| 2nd day | 11 " | 145 " |
| 3rd " | 31.5 " | 174 " |
| 4th " | 37.5 " | 179 " |
| 5th " | 42.5 " | 222 " |
| 6th " | 76 " | 286 " |
| 7th " | 93 " | 338 " |
| 8th " | 100 " | 333 " |

The following table gives average figures derived from various authorities:—

| | Days | | | | | | | |
|-----------------------------|------|------|-------|-------|-------|-------|-------|-------|
| | 1st | 2nd | 3rd | 4th | 5th | 6th | 7th | 8th |
| Schiff | 17.1 | 43.2 | 42.7 | 126.1 | 207.0 | 233.7 | 232.5 | 256.8 |
| Reusing | | | | | | | | |
| (a) Breast-fed | 18.0 | 38.6 | 64.0 | 84.0 | 121.5 | 147.7 | 171.5 | 217.2 |
| (b) Artificially fed | 28.8 | 59.7 | 111.4 | 153.8 | 198.0 | 237.7 | 278.7 | 371.0 |
| Aronstam | 5.8 | 25.2 | 37.4 | 52.0 | 66.5 | 108.0 | — | — |
| Kotscharowski | | | | | | | | |
| (a) Fed by mother | 4.6 | 27.4 | 58.7 | 127.7 | 171.6 | 215.3 | — | — |
| (b) Fed also by other women | 63.1 | 65.8 | 66.5 | 101.0 | 224.0 | 281.5 | — | — |
| Geist | 16.8 | 40.7 | 40.0 | 93.8 | 131.0 | 205.8 | — | — |

The ratio of the amount of urine to the amount of nourishment taken varies according to the absolute amount of the latter. When the intake of fluid is small, the body of the infant is in need of water, wherefore relatively small amounts of urine are excreted. When, however, larger amounts of fluid are taken, as is usually the case with an artificially fed infant, there is an increase both in the absolute amount of urine and in the percentage of the fluid taken in with the milk which is excreted by the kidney. Accordingly, in a case of abundant intake of milk from the third to the sixth day, Cranier found 54 to 60 gm. urine per 100 c.c. milk drunk, Reusing found in the breast-fed infant a percentage of about 22 to 28 during the first four days, whereas, in the artificially fed child, it was 37 to 74 per cent. The percentage in the former from the fifth to eighth day was 44 to 62 per cent., and in the latter 66 to 79 per cent.

The amounts of urine excreted during the first days of life are markedly less than is the case when the infant is older. Urine is frequently passed immediately after, or even during birth. During the first and often also during the second day of life urine is usually only passed at rare intervals: one to two, or three to four times in twenty-four hours. It also happens not infrequently during the first day that a child does not pass any urine at all; this occurs in actually 34 per cent. of all cases, according to Kotscharowski, but is not clinically to be regarded as an alarming symptom. Schiff observed some cases where no urine was passed during the first four days of life, but these were surely examples of early anomalies of excretion of urine. From the third or fourth day of life

onwards the urine excreted begins to increase rapidly in amount, in conjunction with the increased intake of fluid, although urine is still passed less frequently than by the older infant. During the second half of the first week the nappies when changed before each meal are usually found to be already wet, since evacuations usually occur at least six to eight times in twenty-four hours.

The specific gravity of the urine is usually 1006 to 1012 the first day of life; between the second and fourth days it is usually somewhat more, 1008 to 1012, after which it falls gradually to the low concentration characteristic of the normal infant, 1003 to 1004.

The persistence of the high concentration depends on the size of the meals, and especially on the intake of fluid.

The urine passed immediately after birth or during the next few hours usually has a fairly low concentration; it is clear and colourless or of a pale yellow colour. During the second half of the first day it usually assumes the appearance characteristic for the period of the physiological loss in weight. At this time the colour is a fairly deep yellow, sometimes even a brownish yellow, and it becomes cloudy on standing for a short time. This cloudiness is due to the precipitation of the urates on cooling, and is usually of a pale yellow colour and fairly opaque. In the dependent parts of the collecting tubules a dark yellow or reddish sediment is found (urine infest). The urine clears on warming and allows its own colour to be seen. During the second to the fourth days the urine when passed is usually already in a markedly cloudy condition. From the fourth or fifth day onwards, provided that the urine contains enough water, the colour usually becomes paler and the urate cloudiness disappears. With increasing amounts of fluid drunk the urine gradually assumes the almost colourless and watery quality which is characteristic of the normal breast-fed child. When the intake of nourishment and fluid is plentiful this condition may already have been reached by the end of the first week; often, however, the urine remains more deeply coloured until the end of the second or third week.

The reaction of the urine is acid—certainly at the time of highest concentration it is fairly strongly acid, but after the disappearance of the cloudiness the acidity diminishes.

THE ALBUMINURIA OF THE NEWBORN CHILD.

Almost all infants excrete small quantities of albumin during the first days of life. The amount, however, is always exceptionally small, $\frac{1}{4}$ per 1,000 being a maximum which is seldom reached. On applying the acetic acid K-ferrocyanide tests all the albumin present in the urine is usually precipitated. When acid is added to urine which has previously been diluted and cleared by filtration or heating, the intensity of the reaction differs distinctly in individual cases; sometimes it is only a slight opalescence, sometimes a fairly thick, milky cloudiness. Addition of K-ferrocyanide to the filtrate from this produces a fresh cloudiness only in exceptional cases.

Ssesenewski accordingly describes "albuminuria" in the newborn child as a rare phenomenon, while "mucinuria" is an almost invariable occurrence at least during the first days of life. It is now fairly generally recognized that the protein bodies precipitated by the addition of acid are neither mucin nor nucleoprotein. If we are to agree with Mörner in thinking the acetic acid precipitate to be correlated with the presence of substances precipitating (primarily chondroitinsulphuric acid, but also nucleic acid and bile acids), we must then conclude, from the result of this acid test, that the urine of the first days contains relatively large amounts of such substances, in addition to traces. These albuminous substances are even present to excess, as the author is able to affirm, since the addition of a dilute solution of albumin to urine acidified with acetic acid causes a fresh cloudiness to develop. Disregarding the very first amounts of urine, which appear to be absolutely or practically free from albumin, it will be seen that the maximum amount and maximum concentration of the excretion of albumin occur in the first three days of life. Only in about 4 per cent. of the cases observed did the author find the urine entirely free from albumin during the first four days. The reaction for albumin precipitants was never negative during the first three days (cloudiness of the acid urine on addition of albumin being constant). In the following period the albumin reaction tended rapidly to disappear or to show minute traces only. When more highly concentrated urine is passed in the course of a longer period than usual, and when it retains the qualities of infant urine, the excretion of albumin usually also lasts longer. When the urine is concentrated and rich in urates, it usually contains relatively a larger amount of albumin than at any other period, but albumin may also be found before the infant period in the clear urine of the first day. Traces of albumin precipitates are also found during the second half of the first week and not uncommonly still later. In the watery urine of the older suckling, however, these appear to be absent under normal conditions just as is albumin itself.

The albuminuria of the newborn child, together with icterus neonatorum and certain variations in the composition of the urine and of the blood, belongs to that group of phenomena which in older subjects we are accustomed to regard as purely pathological. It is, therefore, considered sometimes from a physiological and sometimes from a pathological point of view. In the latter case it is brought into causal association with infectious and toxic agencies. Among the various reasons given for the excretion of albumin in earliest infancy, the least forced seems to be its association with the circulatory disturbances shown, especially in the congestion of the kidneys, which occur in a more or less marked degree after every birth. That congestion in the region of the kidney can cause albuminuria is known from the pathology of later life. One is here reminded of the orthostatic albuminuria which is now fairly generally recognized as being due to renal circulatory disturbances.

Analogous with this is the excretion of the acetone bodies and of the albumin precipitating substances in the infantile albuminuria. Hofmeier's objection that confinement can have no influence on a process taking place several days later is not valid, since the passive hyperæmia of the internal organs lasts several days, and according to its intensity the excretion of albumin usually shows a marked diminution after three or four days. One may also assume that the process of alteration of the kidney due to the congestion takes a certain time to regain its normal function. The insufficient amount of blood passing through the kidney as a result of the water losses and small intake of fluid at the beginning of life, may influence considerably the course of excretion of albumin. Similarly the infarct of uric acid may be of importance in the ætiology of the albuminuria of the newborn child. All these occurrences may, however, be relegated to the domain of physiology. Nevertheless, it is easy to understand the formation by the physiologically altered kidney of a "*locus minoris resistentiæ*" for pathological influences. The course and intensity of the albumin excretion may certainly be influenced by infective agencies, even by those of the mildest character, such as substances with toxic action derived from the alimentary canal or liberated by the mother. These, however, can hardly be looked upon as primary causes of the very frequent mild albuminuria present in clinically perfectly healthy infants.

OTHER URINARY CONSTITUENTS.

In several cases of children born with the assistance of forceps, Hœniger observed transitory glycosuria only lasting three to four days and gradually diminishing. Since the spontaneously delivered infant does not excrete sugar in the urine even when the labour is of fairly long duration, Hœniger considers that the incidence of the glycosuria must result directly from the sudden force of the operative interference. There are at present no more extensive findings as to the frequency of traumatic glycosuria.

The purely alimentary diabetes mellitus which appears in older diseased infants has not yet been observed in the newborn subject; only in prematurely born children has the excretion of lactose been noted (Nothmann). There are no observations as to the presence of other forms of sugar in the urine of the newborn child (cane sugar, galactose, levulose and maltose).

The excretion of acetone bodies, more especially acetonuria, is well known to occur very readily in childhood as a result of fever and inanition. Small amounts of acetone are also found frequently in the urine of newborn children whose nourishment is insufficient or barely sufficient. The formation of acetone may be recognized easily by the characteristic smell of the breath, but the small proportions of acetone in the blood have merely a symptomatic importance in the above circumstances. Larger excretion of acetone

bodies, especially of aceto-acetic acid and β -oxybutyric acid, is among the rare occurrences of the earliest period of life.

Bile Pigments.—Bilirubin appears in the urine in cases of icterus neonatorum, and is here present not in solution, but as a precipitate (*masses jaunes*). In more severe icterus (e.g., after hæmorrhage, in septic infections, &c.) dissolved bilirubin is also occasionally found. Since processes of reduction are rare in the alimentary canal of very young children and of breast-fed infants, it is not surprising that the products of reduction of bile pigments, urobilin and urobilinogen should be found only in exceptional cases in their urine; nevertheless, the author has already come across some cases in which unmistakable urobilinuria appeared at the end of the first week, accompanied by the excretion of reduced pigments in the faeces.

Intestinal putrefaction in the newborn child is as infrequent as the reduction processes which are its usual concomitants. It is noteworthy, however, that indican is frequently found in the urine during the first week after birth, and this may sometimes occur in considerable amounts without constituting a pathological condition. It may be observed both under conditions which are very good and under those wherein there is insufficient increase in weight; similarly, it may occur both when the intake of food is plentiful and when barely sufficient; it may be accompanied by frequent defæcation or by a tendency to constipation.

Indicanuria is usually absent on the first day, uncommon on the second day, and most frequent and plentiful on the third and fourth day, though it is not unusual to find it during the following days also (v. Reuss, Leo and Passini). If one accepts the parenteral origin of urinary indican, the indicanuria of the newborn child can be looked on as a sign of tissue destruction and ranked with other symptoms of this occurrence. On the other hand, one must not overlook the fact that the indican may possibly be derived from intestinal putrefaction, since the meconium contains putrefactive bacteria (Passini). Dementicell gave albumin water to newborn children, and subsequently found indican in the urine in three cases out of seven. Meyerhofer found one case where glycanonic acid and two cases where indoxyl appeared in the urine on the first day. He also draws attention to an article by Mornadlowski, who detected the presence of indican in the urine a few hours after birth.

Gundobin discusses researches by Kotscharowski and Gein as to the presence of toxic substances, in which both writers conclude that the urine of the newborn child is distinctly more toxic than that of the suckling, while this again is of a higher toxicity than adult urine. From the second to the fourth day the urine possesses particularly toxic properties. Gundobin supposes that this condition of the urine is caused by poisonous substances originating in the tissues, but further studies on this subject are very desirable.

The urinary sediment contains during the infant period plentiful granular and spherical formations, and also rods of amorphous

urates, and crystals of uric acid, sodium urate, calcium oxalate, &c.; while at the time of the icterus the golden yellow granules and flakes of bile pigments are present. Besides the above, epithelial cells are also frequently found, which must come partly from the ureter, bladder and urethra, but partly also from the kidney, further leucocytes and red corpuscles (often lacerated by alkali). The cellular elements are present in many cases, usually in comparatively small numbers. Isolated hyalin casts or cylindroid formations may here and there be seen; these are often encrusted with precipitated urate and are then somewhat difficult to recognize. These bodies are probably due to the same alteration of the kidney which gives rise to the albuminuria, and have no pathological significance.

Digestion

THE PROCESSES IN THE ALIMENTARY CANAL.

If we consider the sensitiveness of the young infant to alimentary injuries, together with the relative ease with which digestive disturbances may result from unphysiological feeding with cow's milk, we are at once faced with the question as to whether the newborn child shows any peculiarities of digestion and assimilation as compared with the older infant. It must first be determined whether differences in alimentary decomposition of foodstuffs may be established between the newborn child and the older infant and differences in the conditions governing absorption.

A large amount of evidence is at our disposal (Ibrahim) as to the digestive ferments of the newborn child.

(a) *Enzymes breaking up proteins.*—Pepsin, as has long been known, is present not only in the gastric epithelium of the newborn child, but also in that of the foetus from the fourth month onwards. According to Rosenstern, the amount of pepsin is somewhat smaller than in the case of the older infant; according to his investigations this rises in amount in the case of the healthy (artificially fed) infant until about the end of the third month, when its amount remains fairly constant. Hydrochloric acid and rennin are also recognizable in the gastric mucous membrane from the first day of life onwards (Szydlowski, Hamburger and Sperr).

Cohnheim and Soether were able to show that newborn puppies had a psychic secretion of gastric juice on the first day of life, through sucking the teats of the mother animal—a reflex action being set up through the receptive organs of the head, thus producing secretion. It is quite permissible without further evidence to assume that this also occurs in man. The act of sucking forms an integral part of the work of digestion.

Trypsin had already been found by older observers in the pancreatic extract of the newborn child. According to Ibrahim, negative results are due to the fact that in the researches concerned the activation of the trypsinogen was omitted. He was able to detect the latter as early as the sixth month of foetal life. Ibrahim deter-

mined the presence of enterokinase, which is necessary for the conversion of trypsinogen to trypsin, both in the full term and in the prematurely born child. It was likewise present in the extracts of intestinal mucous membranes (the lower part of the lower third of the small intestine being the most active), and also in the intestinal contents. According to the researches of Ibrahim and W. Gross, secretin, the hormone causing pancreatic secretion, is also to be found in the extracts of the small intestine of the full-term child at birth. The activity of this is certainly small, so that Ibrahim, who also considered the results of Wentworth's investigations, concluded that the formation of secretin is still in arrears in the newborn child.

Trypsin, the enzyme present in the mucous membrane of the small intestine which hydrolyses albumose and peptone, was found in the newborn and prematurely born infants by Colnheim, Jøeggis, Langstein and Söddin.

(b) *Enzymes acting on Carbohydrates.*—Lactase, the enzyme acting on milk sugar, is present in both the faeces and intestinal contents at birth, and also in the intestinal mucous membrane (Paurz, Vogel, Weinland, Orban, Ibrahim). It is a curious fact that this enzyme does not appear until the later part of embryonic life, thus newborn children from the last month of foetal life are sometimes without the enzyme. Also Nothmann was able to find it in only a few cases in the faeces of premature infants. Under the influence of a diet containing milk sugar the amount and strength of the lactase increases. Nothmann's observations concerning the lactosuria of newborn children shows that the breaking up of lactose may be deficient. It is possible that the formation of the enzyme in the intestinal wall may also be deficient.

It is noteworthy that the enzyme acting on cane sugar (invertin, saccharase) is among the earliest to appear in the embryo (Colnheim, Ibrahim), although under physiological circumstances wherein the infant is fed on milk sugar it is not used for some months. It was detected in the small intestine of the newborn child by Miura. The meconium also contains invertin.

The enzyme acting on malt sugar, maltase, was found by Ibrahim in all parts of the small intestine and in the contents of the same.

The amylolytic ferment (diastase) appears to be present both in the salivary glands and in the pancreas. Ibrahim found ptyalin in both the parotid and the submaxillary glands and also in the saliva. Extracts of pancreas from the newborn child sometimes give positive and sometimes negative results. Ibrahim is of opinion that the pancreas of the newborn child is distinctly slow in assuming its function of producing diastase. The older specialists in children's diseases were therefore right in suggesting that young infants should not be fed on a diet containing much starch, since their advice rests on a physiological foundation.

Enzymes acting on Fats.—Pancreatic steapsin was already

discovered in the full-term infant by Zucifel. His discoveries were supported by Ibrahim and Hartge who also obtained positive results with the fetus. The meconium also contains a fat splitting enzyme. Ibrahim and Kopec were able to demonstrate the presence of lipase, a very powerful enzyme in the gastric mucous membrane of the fetus. Finizio also found that a glycerine extract of the gastric mucous membrane possessed a considerable power of acting on fats.

It thus appears that the infant brings with it into the world the enzymes necessary for the decomposition of foodstuffs in its alimentary canal. The knowledge of this fact has also its practical value, for instance we see that in the diet of a young artificially fed infant it is unnecessary to replace the usual diluted cow milk with mixtures containing predigested proteins. The therapeutic administration of enzymes will thus be useless equally in the newborn child and in the older infant. Should there be a distinct backwardness in the quantity and activity of the digestive enzymes during the first days of life, it will probably be safe to assume, that when nourishment is taken these will soon be formed in corresponding amounts.

Even though the grosser mechanism for alimentary digestion may be in order in the newborn child it is none the less possible that this may show certain differences of method, in both disintegration and more especially absorption, which distinguish it from the corresponding digestive process occurring in the older infant. Various experimental facts indicate that the intestinal wall during the earliest days of life does not offer the same resistance to foreign bodies in the intestinal contents as is the case in the older individual.

Our knowledge as to the permeability of the intestinal wall in the post-natal period is founded on evidence derived from experiments made on animals and it is not permissible to transfer these results directly to man without further evidence. Nevertheless these experiments do throw some light on certain phenomena of this period. What do we understand by the permeability of the intestinal wall? It must not be thought that the intestinal wall constitutes a simple filter, which is, for instance, impermeable to colloidal substances with large molecules but permeable to crystalloids; the intestinal wall, or rather its mucous membrane is a much more complicated functional organ which only acts as a dead membrane to a very limited extent. It is only necessary to refer to the fact that it seems to be impermeable under normal conditions to several crystalloids such as the disaccharides, while the very large molecules formed by the decomposition of proteins can be absorbed. The passage of food materials takes place in one of the following ways: Firstly, the products of decomposition may be taken up by the intestinal cells and passed unchanged into blood or lymph, or decomposition may first take place in the intestinal wall (e.g., in the case of several disaccharides) or finally synthesis

may take place in this locality. The food which leaves the intestine under normal circumstances is by no means the same as that taken up by the intestinal mucous membrane. The permeability of the intestinal wall will therefore have a different significance in each of the following conditions: (1) If constituents of the intestinal contents enter the intestinal wall and pass through it which under normal conditions are not taken up in unaltered condition (this may be a consequence of the coarseness of the "filter" or of an inborn or hereditary cellular insufficiency); or (2) if the intestinal wall absorb constituents of the intestinal contents just as under normal conditions, but passing them on unchanged without accomplishing the splitting up or synthesis which takes place in the normal course of digestion. It is not impossible that the intestinal wall of the newborn child is as backward in the development of its absorptive functions as it is in digestion. The above researches were carried out chiefly in order to determine whether in the newborn, contrary to what occurs in older individuals, antigens on the one hand and protective substances on the other can enter the body by the intestinal tract. Since these substances are intimately connected with the proteins, the latter have been particularly carefully studied. It is asserted on many sides that the milk-protein lactalbumin is able to pass into the blood without hydrolysis, and since it is identical with serum albumin a process of "assimilation" appears to be unnecessary. Bauerstein therefore assumes that the proteins of the colostrum are taken up unchanged by the body through the intestine thanks to the curious anatomical quality of the alimentary canal at this period, and that the organism is supplied with the plentiful, genuine protein complement. A doubt is thrown on the validity of this theory by the fact that it assumes the protein splitting ferments of the alimentary canal to spare the lactalbumin either entirely or to a considerable extent. On the other hand the view that a transit of the protein substances does actually take place is supported by successful demonstration of the passage of antitoxin through the intestinal wall when the latter is incorporated with the similar lactalbumin, homologous with the infant. It is shown by numerous researches that antitoxins, haemolytic antibodies, agglutinins, &c., are able to pass through the intestinal walls of the newborn child, although perhaps not in the case of all animals; this, however, does not take place in the older individual (Römer, Ganghofner and Langer, Uffenheimer, Hamburger, Bertarelli). These bodies also seem to pass through when incorporated with foreign proteins in the intestine. This permeability of the intestine for foreign protein bodies of other species of animals forms a very significant contrast between newborn and older individuals; according to Hamburger the intestine of the latter has the function of protecting the organism against the invasion of foreign proteins. Ganghofner and Langer give the seventh day as the latest time at which proteins can pass through in animals (dog, cat, rabbit, kid); they found a similar condition in the human infant but conjecture

that the intestine of the latter may only be permeable during the first four days of life.

The practical importance of this increased permeability of the intestinal wall of the infant is obvious; on the one hand there are its advantages (absorption of the protein of the colostrum and of protective substances from the milk of the mother), on the other hand are disadvantages (formation of antibodies, on the part of the intestine when foreign proteins are present in the food, entrance of toxins and toxic products of digestion, &c.).

We have but little information in respect of the fate of the digestive products absorbed from the intestine and the specific processes in their intermediate metabolism. It is as yet undecided whether there is a paraportal absorption, an absorption through the ductus venosus Arantii with circumvention of the liver (Römer, Gessener) or if present whether such a process is of any great importance. The obliteration of the ductus venosus is usually not complete for two months (Haberda), but the process of obliteration begins as early as the first few days of life, and although it may be possible to fill the duct with injected fluid right to the vena cava in the bodies of infants a few weeks old, the actual blood flowing through it is probably small. It is possible that the liver itself may show a certain insufficiency of function, so that the products of intestinal digestion flowing to it in a perfectly normal way are not further acted on as they would be in later life. Gundobin, for instance, attributes the relatively high excretion of ammonia during the first days of life to a deficiency in the "oxidizing function" of the liver, meaning that the most important functions of the liver, from the qualitative standpoint, are the same as in the adult, but not yet fully developed. It is possible that the function of the liver in removing poisons is also deficient. It is, however, unnecessary to go as far as Gundobin, who assumes that the newborn child, chiefly on account of the incompleteness of its metabolism, is in a state of mild toxæmia; under physiological conditions especially, that is, where nourishment is with the milk of the mother, the functional activity of the intestines and the process of intermediate metabolism show themselves to be quite adequate. When, however, as a result of abnormal conditions in the alimentary canal, due to disease of the intestinal wall or to unphysiological feeding with cow's milk, &c., divergencies occur from the normal infantile processes in the lumen and walls of the intestine, a disproportion between the working and the functional limits of an organ may easily arise. We now have the explanation of the fact that injurious influences arising in the digestive tract are as a general rule followed by more serious consequences in the first few days of life than in later infancy.

DEFECATION IN THE NEWBORN CHILD.

The intestinal contents, which have been formed from the fourth month of foetal life onwards, are known as meconium. This is

excreted in the course of the first days of life by peristalsis which is activated by the birth process. Besides the secretions of the fetal alimentary tract it contains shed epithelial cells and swallowed amniotic fluid. The meconium excreted during the first days is blue-black, tenacious, sticky, homogeneous and without smell. Sometimes the first portion excreted—the so-called meconium embolus—has a grey white or yellowish glassy appearance, and consists of secretions from the lowest part of the intestine, of mucus and cellular elements (see p. 248). The dark green meconium coming from the large intestine is usually followed on the second day by brown-coloured masses; the transition from the green (meconium hepaticum of Huber) to the brown (meconium amnioticum) is formed by excretions of an olive-green colour. This is followed, first by dark brown, then yellowish brown, and later strikingly bright-coloured intestinal contents still showing the consistence of meconium.

Different figures have been given for the amount of meconium. Cramer gives 60 to 90 gm., others give much higher amounts (see p. 3). It is not easy to determine the end point of the excretion of meconium; the change to the "starvation faces" (in cases of delayed nourishment) or to the so-called "transition faces" is gradual. According to Berster, who examined the excretion of meconium in seventy-four newborn children, the time of its disappearance lies between forty-eight and ninety-six hours. The number of evacuations is variable, for they occur one to three times daily in large single portions or in numerous smaller amounts.

The characteristic colour of meconium is due to bile pigments, bilirubin and biliverdin both being present. Other constituents of bile, especially bile acids (taurocholic and glycocholic), are also present in fairly large quantities. The nitrogen content of the meconium amounts to $2\frac{1}{2}$ to 5 per cent. of the dry residue. The ether extract (15 per cent. of the dry residue) contains, in addition to the pigments and unknown bodies which form its main bulk, a small proportion of fatty acids and fat, the fatty acids in the meconium being similar both in melting point and iodine value to the fatty tissues of the newborn child (Knöpfelmacher). Besides these cholesterol is present in the meconium, but stercorin is absent. In some researches leucin and tyrosin were found. Under normal conditions products of putrefaction and reduction do not occur in the meconium, which is free from bacteria during the first hours after birth, and is usually poor in bacteria later; as a result of this, neither phenol, indol, nor hydrobilirubin are found. According to Schmidt, "there is no fluid or semi-fluid constituent of the human body which resembles the meconium in its power of remaining many months either inside or outside the body in a practically unaltered and undecomposed condition. Meconium only becomes offensive when moistened with water, when it becomes so quickly (Czerny-Keller). Not infrequently meconium gives the reactions for blood pigments, and this is probably due to the maternal blood swallowed during labour.

somewhat greenish, showing small light yellow fragments of soap. Generally the first symptom which makes the "milk stools" recognizable as such is the extraordinary and characteristic sour smell. The yellow colour rarely appears before the fifth or sixth day, and then only when the intake of milk is plentiful. The normal "milk stools" of the first period almost always contain mucus and are friable. Pasty, homogeneous stools are exceptional; they often appear in somewhat overfed infants and not infrequently alternate with their watery evacuations. (For further details, see p. 220).

The first bacterial infection of the intestine, according to Escherich, occurs as early as three to seven hours after birth, but Tissier, taking two-hourly samples of meconium from the rectum, gives the tenth to the twelfth hour as the time of the first entry of bacteria. According to Sittler, this usually occurs about the twelfth hour after birth. In his opinion the infection of the intestine begins with the appearance of solitary specimens of enterococcus, which later occur in groups together with a few specimens of *B. coli*. During the next twelve hours these germs increase but slightly in number, but there appear a few other species of anaerobic bacteria of the class described by Gruber and Schottenloeh as "immotile, dimorphic *B. butyricus*," also the *B. perfringens* or "Gasphlegmone bacillus." According to Passani, it is probable that other bacteria are also concerned in the formation of the meconium bacilli; the *B. perfringens* is, however, the one which is chiefly concerned in forming the characteristic "meconium flora." This bacillus shows a fairly distinct polymorphism, appearing, on the one hand, either as the so-called "drum-stick" form, which resembles in appearance the "Köpfchenbacteria" of Escherich (oval spores with long, thin threads) or as thicker rods with terminal spores (forms of clostridium), and, on the other hand, as long, thin, thread-like forms. The *B. perfringens* stains positively with Gram, but this is by no means always clearly marked, and sometimes it does not stain at all, or shows unevenly stained, streaky bacilli, which are evidently dying.

The further history of the bacterial infection of the intestinal contents is given by Sittler as follows: The thread-like variety of the *B. perfringens* grows more rapidly than the other two forms, and reaches its maximum development at the end of the second or beginning of the third day. The *B. bifidus*, which permanently remains the chief bacterium of the stools, begins to develop rather immediately after the disappearance of the *B. perfringens*, or after a temporary increase in the number of the enterococcus present. Before the appearance of the *B. bifidus*, especially during the transitional period of the stools, the already present "Gasphlegmon bacillus" may change into its spore-containing form; the bacillus appears to be able to sporulate in the meconium only in the presence of *B. coli*. The transition from this flora to the permanent bifidus flora then follows in the manner described above, either directly or with an intervening period wherein the

growth of enterococci preponderates. (These latter appear at the same time when thin dyspeptic stools are excreted; i.e., when the normal flora of the small intestine have passed down to the colon.)

According to Sittler, the *B. acidophilus* does not appear until after the *B. bifidus*, in the middle of the fifth day of life at the earliest. From about the fourth to the sixth day the stool flora have usually assumed that character, which they also present later, consisting of an almost pure culture of *B. bifidus*.

It is not yet certain whether the first infection of the intestine chiefly occurs from the anus (Escherich, Passini) or through the mouth (Tissier, Sittler). The fact that a number of intestinal bacteria (enterococcus, *B. coli*, *B. perfringens*) also occur in the buccal cavity of the newborn child might possibly indicate the especial importance of oral infection. They reach the mouth chiefly from the generative organs of the mother. Infection from the air and from the bath water can only be of subordinate importance. Nevertheless the conditions of environment are certainly not without influence in this connection, whence Passini observes that the meconium of twenty-four-hours-old infants from a certain maternity clinic was still almost entirely free from bacteria, while in another institution a large number were present. A further source of infection for the alimentary canal is the breast of the mother, both the nipples and the glandular secretion itself. Since micro-organisms are present both on the outer skin and in the ducts of the mammary gland—cocci, *B. acidophilus* (Moro), &c.—this mode of infection may be of importance in the spreading of infection in the intestine. The origin of the *B. bifidus* is still obscure.

(C) THE BLOOD

The composition of the blood of the newborn child shows several peculiarities, both morphological and clinical, as compared with that of older infants.

The majority of writers agree in giving the hæmoglobin content a remarkably high value during the first days. E. Schiff has made the following table:—

| | Prague | Budapest |
|---------|----------------|----------------|
| 1st day | 124% per cent. | 122% per cent. |
| 2nd " | 104.2 " | 135.0 " |
| 3rd " | 110.1 " | 135.0 " |
| 4th " | 96.5 " | 130.2 " |
| 5th " | 94.0 " | 132.5 " |
| 6th " | 94.5 " | 127.7 " |
| 7th " | 92.5 " | 120.8 " |
| 8th " | 90.7 " | 118.1 " |
| 9th " | 96.3 " | 115.1 " |
| 10th " | 96.3 " | 110.6 " |

According to Gundobin, the hæmoglobin content amounts to 90 to 115 per cent. in the newborn child. Trump gives 110 per cent. and more. Takasu found values of more than 120 per cent. in the first days in the majority of Japanese infants. It may be deduced from these results, and also from Schiff's tables, that racial peculiarities play a part. Heilmann found the hæmoglobin content of the blood of jaundiced children to be distinctly lower than that of normal infants. As a general rule it may be considered that the hæmoglobin content begins to fall from the very day of birth or after a slight rise of three or four days' duration. The fall is regular, and the value normal to the infantile period (about 70 to 80 per cent.) is reached in the course of the second or third weeks.

The number of red blood corpuscles is relatively large in the newborn child, for there are, as a rule, more than 5,000,000 per c.c., and lower values are rare. Sometimes more than 8,000,000 may be counted. In severe cases of icterus Heilmann found lower values—5,500,000, as against 6,500,000 in non-jaundiced infants. Gundobin found an average of 6,700,000 with variations between 7,500,000 and 5,000,000. Scribnier gives an average value of 6,980,000, and Biffi or Galli one of 7,000,000. Further high values are given by Perlin (6.1-6.2 millions as average from the second to the fourth day), Fehrsen (over 6 millions on the first day), Bidone and Gardini (6 millions). Takasu gives as average value only 4,690,000, but his estimates do not deal with infants of less than four days. According to Viereck, the blood of male infants is richer in erythrocytes. The maximum count is usually reached on the second day, less frequently on the third or fourth day. It then falls gradually, and reaches an average of 4½ millions by about the third or fourth week. According to Heilmann, in jaundiced infants the hæmoglobin content, the red corpuscles and the specific gravity all increase from the third to the fourth day, while in the case of non-jaundiced the values fall from the third day. According to Schiff, the time of ligature of the umbilical cord influences the number of red corpuscles inasmuch as when it is done late the values rise until the second to the fourth day, and when early the fall begins immediately.

The qualities of the red blood corpuscles of the newborn child may be summarized in the following manner (Gundobin):—

(1) The size of the red blood corpuscles shows marked variations—between 3.25 and 10.25 μ —the large ones being larger and the small ones smaller than in the adult (Hayem).

(2) The physical characteristics of the red corpuscles differs from those of the adult; they take up fluids more readily, and are more easily hæmolyzed in this way or by reagents; small cells readily assume a spherical form (Hayem). Hofmeier saw incomplete nummular formation quite frequently. Fischl and Heymann but rarely.

(3) The hæmoglobin is not over firmly attached to the red

corpuscles, which accounts for the presence of a larger number of "ghosts" (Salbermann, Scherenziss).

(4) The red corpuscles contain more stroma (Scherenziss).

(5) In the course of the first days nucleated red corpuscles are frequently seen (Wojno-Oranski), being especially frequent in newly born babies (de Vacaris). Carstanjen saw them during the first three and Scribades during the first five days.

(6) Microcytes are much more frequently present in the blood of the newly born than in that of older infants (Hock).

(7) The twenty-four-hourly variations in the number of red corpuscles are very great in the newborn child.

Cuthala and Darnay found, in blood taken from the umbilical cord at the moment of birth, fairly numerous finely granulated erythrocytes (*hématies granuleuses*); these become still more plentiful in the following hours, though after the first day their number decreases, while from the fifth to seventh days they are rare, and after the eighth day exceptional. According to Sabrazès and Leuret, the jaundiced newborn child has three times as many finely granulated erythrocytes as the non-jaundiced infant; essentially they are nothing more or less than the well-known polychromophil red corpuscles (Lehndorff).

The resistance of the red blood corpuscles is discussed on p. 66.

The number of leucocytes is always large in the post-natal period. One can properly speak of a leucocytosis of the newborn child, although the average values given by different authorities differ very considerably, for hardly any of them give figures less than 19,000 or three times the normal value for later life.

| | | |
|----------------------------|------------------------------|---------------|
| Hayem | in the first 24 hours | 18,000 |
| Otto | ... | 25—24,000 |
| Schiff | in the first 24 hours | 14—16,000 |
| Cadet | ... | 19,400 |
| Zangemeister and Meissl | ... | 11,430—20,010 |
| Scribades | ... | 19,268 |
| Peelin | on the first day | 17,146 |
| Fehrsen | on the first day | 7,600—12,500 |
| Takase | from the first to fourth day | 12,000—28,000 |
| Birnbaum | soon after birth | 20,000 |

The leucocytes usually increase in number during the hours immediately following birth, thus Wojno-Oranski found an average of 16,980 just after birth, (twelve hours p.p. 20,080, on the second day 25,580 leucocytes. From this period, however, the number of leucocytes diminishes fairly rapidly. A few authorities, however, have been able to detect a slight rise occurring somewhere between the third and seventh day, but the number of leucocytes at the end of the first or beginning of the second week is usually already between 8,000 and 12,000. The following average values show the changes in the number of leucocytes during the first week:—

| | | | |
|-------------------------------|---|---|--------|
| (A) SCHMIDT'S : | | | |
| 1st day | — | — | 25,208 |
| 2nd " | — | — | 14,248 |
| 3rd " | — | — | 60,734 |
| 4th " | — | — | 9,104 |
| 5th " | — | — | 8,893 |
| 6th " | — | — | 9,490 |
| 7th " | — | — | 10,354 |
| 8th " | — | — | 11,000 |
| 9th " | — | — | 10,335 |
| 10th " | — | — | 9,150 |
| (B) GUNDOBIN'S : | | | |
| Blood from the umbilical cord | — | — | 18,000 |
| Immediately after birth | — | — | 18,000 |
| 6 hours p.p. | — | — | 22,000 |
| 24 hours p.p. | — | — | 23,000 |
| 48 hours p.p. | — | — | 10,000 |
| 5 days p.p. | — | — | 8,100 |
| 7 days p.p. | — | — | 11,000 |

The leucocytosis of the newborn child is due to the polymorphonuclear corpuscles. Carstanjen has set forth as follows the result given by his researches on the percentage proportions of the various kinds of leucocytes present during the post-natal period: At the time of birth and during the first twenty-four hours the blood is very rich in polymorphonuclear leucocytes and poor in lymphocytes. After the first day the number of polymorphonuclear leucocytes diminishes, while that of the lymphocytes increases. Between the sixth and ninth day p.p. the two kinds are present in approximately equal quantities. From then onwards the number of polymorphonuclear leucocytes falls still further, while the lymphocytes continue to increase. Towards the twelfth day it is usual to find already the proportion characteristic of the first month of life, namely, a relative lymphocytosis. In the average curve made by Carstanjen the polymorphonuclear cells show a decrease to 73 per cent. on the first day to about 66 per cent. on the third, and to about 42 per cent. on the sixth day; by the ninth to twelfth day it is already down to about 36 per cent. These results agree in essentials with those of other authorities. Pittaluga gives for the first hours after birth a somewhat lower value for the polymorphonuclear cells (50 to 60 per cent.).

The transitional forms are, according to Carstanjen, relatively numerous in the newborn period, especially so between the sixth and ninth day. With regard to the eosinophil cells the results given are not in close agreement. Carstanjen found that they were not increased in comparison with the blood of older children, also Gundobin gives 1 to 3 per cent. as the normal value. According to Warfield, the percentage of eosinophils varies within wide limits. Heimann finds on the average that non-jaundiced infants have twice as many eosinophils as jaundiced (7 to 8 per cent., as against 3 to 4 per cent.). Myelocytes and mast cells are rare and present in small numbers (Warfield).

Arneth has studied the proportions of the neutrophil polymorphonuclear leucocytes, using his well known method of

arranging the cells, according to the number of their nuclei and loops, into five groups. While the normal adult blood shows a prevalence of nuclei with two to four parts and a scarcity of nuclei with one to five parts, Arneith found on the first day of life a blood curve pushed far to the left, i.e., in the direction of the young cells, the abundance of young cells and the scarcity of old ones being specially marked on the fifth day. In testing these results Heimann was never able to confirm this "movement towards the left."

Reboudi and Morse have made researches on the blood platelets. The former gives the number of 95,000 platelets per cubic centimetre in the newborn child (as against 300,000 in the adult). The latter found immediately after birth either very high (412,000) or very low values (100,000); in the course of the first days of life these differences are somewhat reduced, consequently after the end of the first week the number of platelets varies between 350,000 and 400,000. In cases of *icterus*, especially when it is subsiding, the number of platelets may rise to a million.

According to Schiff the specific gravity of the blood of the newborn child differs individually between 1.080 and 1.060; during the first six days the higher values preponderate, and between the sixth and tenth days the lower ones. The average specific gravity gradually diminishes from 1.0760 to 1.0652 on from the first to the tenth day. According to Karnitzki, the specific gravity of the foetal blood at the moment of birth amounts to 1.0616, it is similar to that of the adult male, and somewhat higher than that of the pregnant woman.

The dry residue of the blood during the first ten days amounts to 21.4 to 27.7 per cent., the ash content 0.79 to 1.34 per cent., the protein content 17.3 to 27.4 per cent. The dry residue diminishes gradually until the tenth day, and chiefly after the first day. The ash content falls until the third day, rising again until the seventh day, falling again from that time. The albumin content varies according to the time of ligature of the umbilical cord. In cases in which ligature has been performed without delay the highest values also occur on the earliest days, and a gradual fall takes place until the tenth day; in cases of delayed ligature the original value rises gradually until the third day, and does not fall till after that time (Schiff).

According to Banerisen, the albumin content of the serum usually falls during the earliest days of life, similarly to the body weight, from 6 to 7 per cent. down to 4 to 5 per cent.; this may be due to a utilization of body protein. Only on rare occasions does it rise while the body weight falls. After the fourth and fifth day the body weight and the protein content of the serum undergo a simultaneous increase. E. Reiss found 6.7 per cent. of protein in the serum of a child one and a half days old and 6.2 per cent. on the seventh day.

The other chemical peculiarities of the blood of the newborn

child are described in the following way by Karnitzki: The iron content of the blood amounts to 0.0512 per cent. and is greater than that of the mother. (According to van Vye, this value varies about 0.045 per cent. in the full-term infant.) The proportion of insoluble salts, 0.3551, is twice as high as in the blood of the adult, though the plasma contains about the same amount as adult plasma. The average content of bases (K and Na) is smaller, and that of chlorine greater, than in the adult. The foetal blood is richer in Na and markedly poorer in K than the adult. The total bases (Na and K) uncombined with Cl is less in the foetal blood than in the adult.

The alkalinity of the foetal blood, according to Ubbel, is similar to that of the maternal, which is diminished during pregnancy. Pfaunder found a distinctly low alkalinity in the blood of weakly, prematurely born infants. He thinks that there is a connection between the low resistance of prematurely born infants against septicæmic and other infectious diseases and the low OH ion content of the blood, since it has been shown that when the alkalinity of the blood is raised the resistance against bacterial processes is increased.

Concerning the osmotic condition, Kroenak and Firth came to the conclusion that maternal and foetal blood are in osmotic equilibrium at the time of birth. The freezing point of these kinds of blood is on the average higher than that of the normal adult (0.520° as against 0.550°); Marbes found only slight differences between maternal and infantile blood, and the experimental results are often very contradictory (Heymann).

The viscosity value of the blood of the newborn child is strikingly high, being usually higher than 6.0 (Trumpf); Amerling gives an average value of 6.7 per cent. It usually exceeds that of the mother by one-third. The Hess hb. viscosity coefficient of newborn blood is usually near the lower margin of normality, and is frequently even lower than this. Trumpf believes that this high viscosity is due to the accumulation of CO₂ and the consequent passage of viscous substances—as well as hb.—from the erythrocytes into the plasma; the blood has a markedly dark-red colour. The CO₂ content of the blood, according to Rielander, amounts to 37.1 per cent. when the umbilical cord has been ligatured without delay, and to 40.9 per cent. when this is done late, and it is noteworthy that in prematurely born and asphyxiated children it is relatively higher. Thus cyanotic newborn infants show the highest viscosity value (12.0, Trumpf). Riesz, on the other hand, explains the high viscosity of the blood as follows: The customary method of ligature of the umbilical cord results in an excess of about 50 to 100 gm. of blood. With this excessive quantity of blood the passage of plasma out of the circulation takes place more quickly than the destruction of the superfluous corpuscles, so that during the first days the blood is always richer in cells and therefore in those constituents which are of primary importance in determining the viscosity, which therefore shows a rise during the physiological loss in weight. Since, during this period, the blood is involved in the general water loss of

the organism, the viscosity is raised still further, but as soon as the weight increases again the viscosity diminishes.

The serum of the newborn child has been studied by the refractive index method, which consists in the determination of the refractive index of a fluid and permits of conclusions with regard to the concentration of cells. By this method it has been shown that the curve of the refractive index represents an inverted image (*Spiegelbild*) of the weight curve. In the first days a rise in the refractive index of the serum takes place and the blood loses water. At that time when the weight curve remains steady the greatest degree of water shortage is observed and when the infant regains its initial weight the serum also regains its original water content (Rott). Until then, however, the curve of the refractive index also falls. If the weight rises uniformly during the weeks following the recovery of the birth weight, one finds once more a slow rise in the refractive index curve, as representing the gradual fall in the water content of the organism as age increases.

The peculiarities of the blood of the newborn child and the modifications which it undergoes in the course of the first week—together with the aforementioned conditions as to viscosity and refraction—are partially, but by no means completely, explainable as being due to alterations in the concentration. It is impossible to observe a close relationship with the physiological loss in weight either in the case of the specific gravity, the dry weight, the ash and protein contents or concerning the morphological conditions. Certain variations which occur during the first days of life, such as the increase in numbers of erythrocytes and leucocytes, may be explained as signs of the increased concentration. We see, however, that the number of blood corpuscles is much greater on the first day than on those days wherein the birth weight, and correspondingly the water content of the first day have not yet been regained; we see, moreover, that during the loss in weight the protein content of the serum falls. These facts lead us to conclude that a utilization takes place of both the corpuscular elements and the dissolved constituents of the blood. The large amount of pigment in the faeces, and perhaps also *icterus neonatorum*, are to be regarded as due to a utilization of red corpuscles, just as the infarct of uric acid and the relatively high excretion of uric acid indicate the destruction of leucocytes. The blood findings agree with the results of experiments on metabolism in showing that during the first days a destruction of tissue takes place, which must certainly not be looked upon as a pathological occurrence, but as a peculiarity of this period of life. The newborn child brings with it into the world a reserve depot which acts as a compensation for the physiological underfeeding of the first days of life.

The leucocytosis of the first days of life is explained in several ways. Rieder thinks that the great changes taking place in the body of the child after birth, such as the alteration of the circulation and condition of the blood, the unusual requirements of the alimentary

tract, &c., produce an increase in the number of leucocytes. Schiff assumes an inflow of lymph into the blood after the intake of food; an aetiological significance is also attributed to the state of starvation of the first days. Against this is the fact that the excess of polymorphonuclear cells is brought into the world with the infant, and therefore must be continued from the intra-uterine period. Arneth explains the changes in the number of leucocytes, during the first days of life, by looking upon the whole process as an expression of the great demands put upon the leucocytes at the moment of birth and also in the following period: "These, even if present in large numbers at birth, have many of their young members absorbed during the first days by the institution of the functions of all the organs, of the tissue metabolism, &c., consequently their number becomes smaller, the older cells rarer, and, to compensate for this diminution, there takes place an increase in the activity of the blood-forming organs. In the case of neutrophils, after preliminary increased production of predominantly young cells, the body of the infant standardizes its activities with regard to the numbers of the cells formed, and consequently the composition of the blood is regulated."

(D) CIRCULATION AND RESPIRATION

While the change from placental to intestinal nutrition is gradual, in so far as the latter only reaches its full operation after some days, the organism meanwhile being forced to supplement the requirements of its metabolism with reserves from the foetal period, the alterations in the interchange of gases takes place with great rapidity. The respiration begins as soon as the placental circulation is interrupted. Although residues from the foetal circulation may persist for a time in extra-uterine life (since obliteration of the foetal communications is gradual), and although the first inspiration does not lead to the entire filling of the lungs, the conditions of the circulation and respiration, at all events from the clinical point of view, are not essentially different during the first days of life from those of later infancy.

Electro-cardiographic investigations show that there are certain differences in the functioning of the heart; the electro-cardiogram of the newborn child is characteristic in form in that it shows an especially deep Jp notch (Hensbner, Furaro, Nicolai). Hecht determined the ratio of the Jp notch to the J notch in a large number of newborn infants; he came to the conclusion that in these the Jp notch is the greatest, being more than three times as large as the J notch; in the course of infancy it hardly exceeds the J notch by half, and in later childhood, apparently independent of age, is on average half as large as the J notch.

The interval between the auricular and ventricular systole

increases during infancy up to the time of puberty; according to Hecht, it amounts to 0.10 sec. in the newborn (in infancy it is somewhat less, and in the first year of childhood 0.13 sec.).

Concerning the physical signs of the heart it has been shown that in infancy the apex beat is normally to be found in the

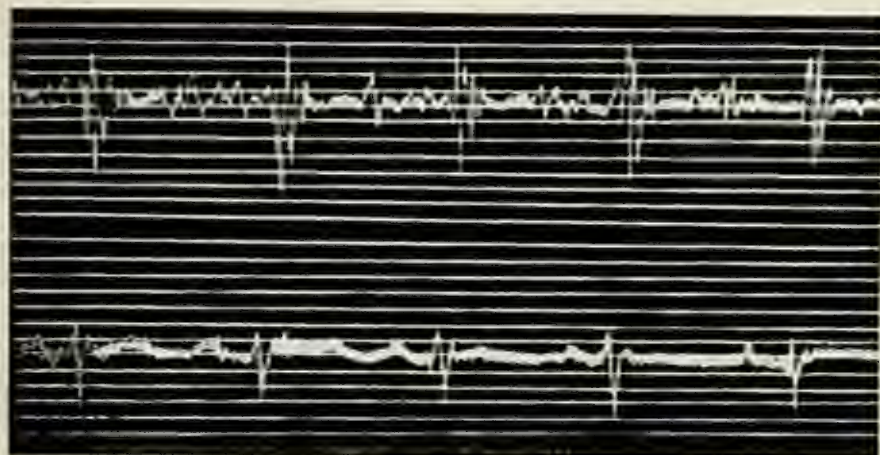


FIG. 7.

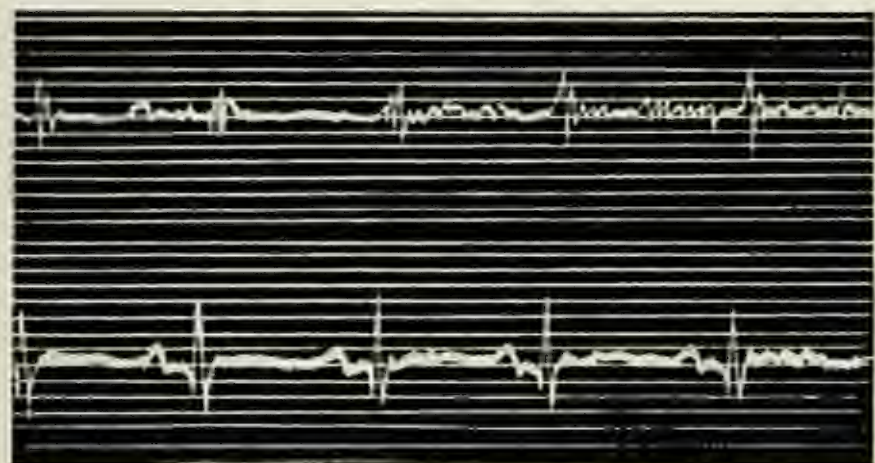


FIG. 8.

third intercostal space, $\frac{1}{2}$ to 1 cm. lateral to the nipple line. This is due on the one hand to the peculiar shape of the infantile thorax, the transverse and deep measurements being approximately the same, and on the other hand to the physiologically high position of the diaphragm in infancy which leads to the heart's being farther to the left and to a more horizontal direction of its

axis than is the case in the adult. Hochsinger has called attention to differences between the early and late infancy as shown by auscultation; in early infancy a trochaic rhythm is present at the base of the heart instead of an iambic. Hecht has now been able to prove indisputably, using Edelmann's "lateral galvanometric

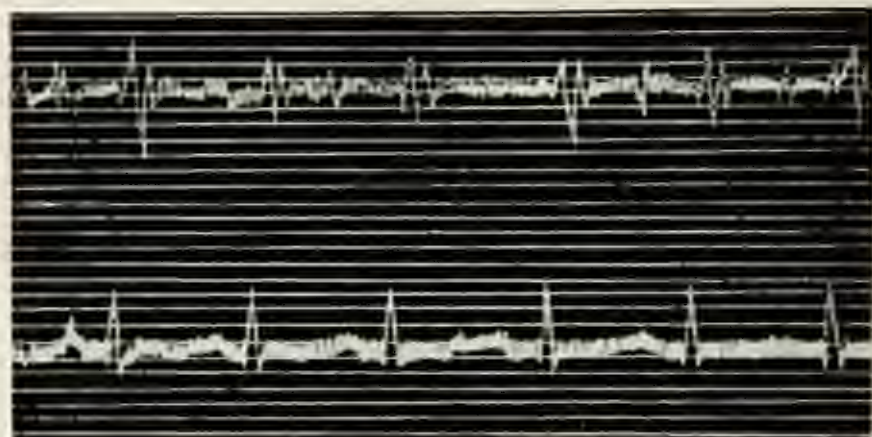


FIG. 9.

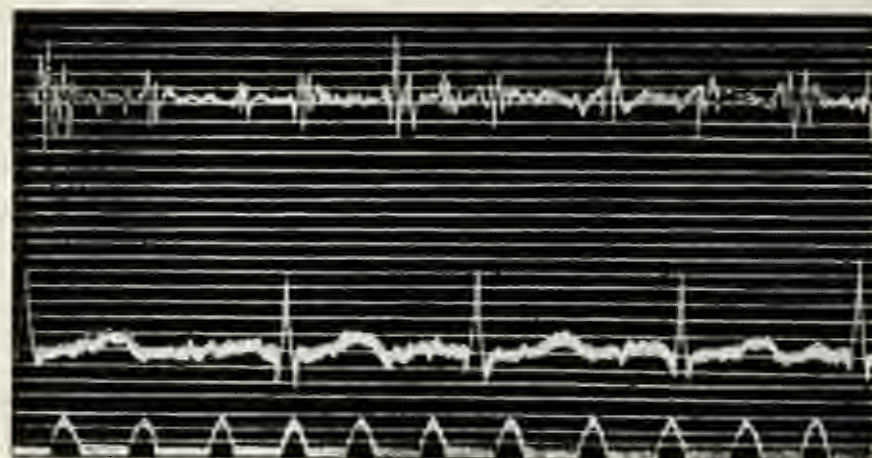


FIG. 10.

registration" (*Seitengalvanometerregistrierung*) of the heart sounds, that the amplitude of the first heart sound is actually greater than that of second sound in the newborn, in the infant, and in early childhood. That Hochsinger's phenomenon is not generally recognized is probably due to the fact that in auscultation one is liable

to hear the rhythm one wishes to hear or is used to hearing, and that is the umbilic rhythm at the base of the heart.

The curves shown are from Hecht, and show these conditions very clearly (figs. 7 to 13). At the lower margin of the tracing the time is shown ($\frac{1}{2}$ and $\frac{1}{10}$ sec.). Above the time tracing is the

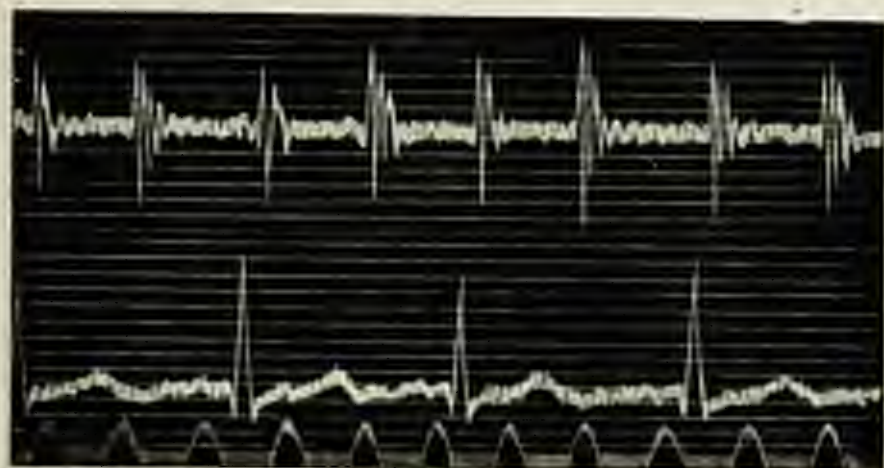


FIG. 11.

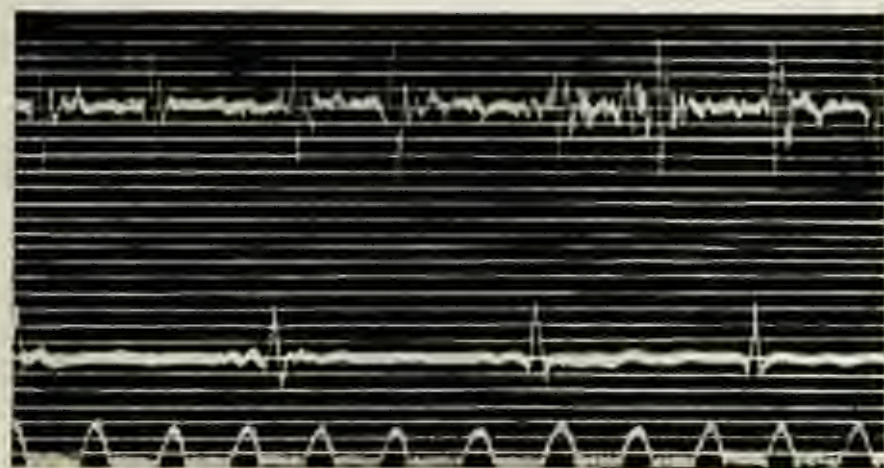


FIG. 12.

electro-cardiographic curve whose high spike corresponds to the beginning of the ventricular systole. According to Kan, the first heart sound begins 0.028 sec. after the beginning of the ventricular spike; and since, moreover, it appears at the moment when the greater part of the cardiac muscle has entered into con-

traction, the first heart sound can be recognized immediately. The second heart sound begins, according to Kan, 0.031 sec. after the end of the final movement, but this is not clearly recognizable in all curves.

Figs. 7, 8 and 9 illustrate the apical (7), pulmonary (8), and aortic (9) sounds of a newborn boy. Fig. 7 shows very deep Jp notch, fig. 8 deep Jp and Ja notches. The subsequent oscillation is indistinct in all three curves, and also the second sound in figs. 8 and 9 is not clearly recognizable over the pulmonary artery and aorta. Figs. 10 (apical), 11 (pulmonary), 12 (aortic) and 13 (tricuspid) are curves derived from an eleven-year-old boy, and serve as a comparison with the former curves. It appears that in the newborn the first sound preponderates not only at the apex but also at the base of the heart, while in the older child this is only the case at the apex and over the tricuspid valve.

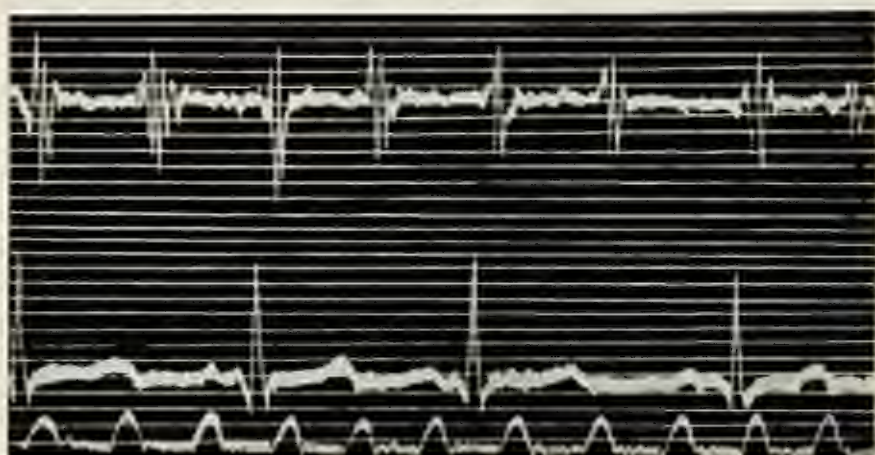


FIG. 13.

The pulse-rate in the newborn is, if possible, still more variable than in later infancy. If investigations be carried on while the child is asleep the pulse-rate will not usually be found so relatively rapid. Hecht gives the number 120.6 as arithmetic mean of the pulse-rate of twenty-seven children of the first week; the individual numbers given by him vary, however, within the very wide limits of 90 and 200. According to Pokrowski, the pulse-rate varies between 98 and 140 during the first days of life, the average being from 100 to 120 beats, which is also lower than the average value of 134 given by Vierordt for the suckling. According to Gundobin, the readiness with which the pulse becomes irregular is due to the fact that the cerebral centres controlling the activity of the heart are as yet imperfectly developed. The following observations of Bouchat serve as an example of the extent to which the pulse-rate may vary in infants at various periods:—

| AGE | PULSE RATE | |
|--|------------|------|
| | Max. | Min. |
| Fetus shortly before birth | 160 | 108 |
| First minute after birth | 164 | 72 |
| First day of life | 135 | 95 |
| Following days until the end of the third week | 164 | 146 |
| Second month to end of second year | 150 | 94 |

The physiological (respiratory) arrhythmia is comparatively slight in the suckling and the young child. The differences between the smallest and largest pulse period, which can be used to measure the intensity of this arrhythmia, increases with age up to puberty; only in the first period of life does it show a small diminution in so far as it is slightly greater in the newborn than in the older infant. Hecht gives the following average values (in $\frac{1}{20}$ sec.):—

| | |
|--------------------|------|
| In the newborn | 3.4 |
| " suckling | 2.8 |
| " prematurely born | 2.5 |
| " young child | 2.25 |
| " older child | 2.4 |

The blood-pressure is low during the first days of life and certainly the lower it is the poorer is the development of the child. Sładko obtained the following figures working with Gartner's tonometer:—

| | |
|---------|---------------|
| 1st day | 62—64 mm. |
| 2nd " | 62½—64½ mm. |
| 3rd " | 63—68 mm. |
| 4th " | 62—70 mm. |
| 5th " | 62—74.7 mm. |
| 6th " | 73.5—78.7 mm. |
| 7th " | 74—78.8 mm. |
| 8th " | 70—80 mm. |

After the blood-pressure has risen gradually during the first week of life it remains practically stationary during infancy.

Tripp found no difference in blood-pressure between newborn and older infants; it amounted on average to 80 mm., and he only found readings of 60 to 70 mm. in prematurely born and weakly children. In *icterus neonatorum* the blood-pressure appears to be raised in the first stage but lowered when the condition is fully developed.

There is a high average frequency of respiration in the newborn; according to Gundobin it amounts to 40 to 50 in the sleeping child, according to Dohrn 50 (during the first ten days of life). Inspiration is less frequent while the newborn child is crying than in usual regular respiration. The depth of respiration from the first to the tenth day increases in such a way that the performance of an expiration on the tenth day (45 c.cm.) exceeds that of the first day by about 12 c.cm. Dohrn sees the explanation of this increase in the greater air-requirement, in the facilitated movement of the costal cartilages and in the increasing patency of the

branches of the bronchi. The depth of respiration is remarkably low in the first hours and remains so throughout the first day. It then rises greatly from the first to the second day, correspondingly the gaseous exchanges begin to rise quickly during the second day (Scherer) and increase slowly during the following days. It thus appears that under perfectly normal conditions a complete filling of the lungs by the first inspirations cannot be effected.

The type of respiration in the newborn is mainly diaphragmatic, as is generally the case in early childhood. The respirations are, as a rule, fairly superficial and irregular. Eckerlein was able to determine the following types of respiration: in sleep expiration and inspiration take place as one process, but different quantities of air are taken up in the individual respirations in this case also. Inspiration and expiration succeed one another rapidly as a rule, inspiration usually taking place more rapidly than expiration; this condition is especially noticeable in the waking state under quiet conditions, a very protracted expiration occurring after a comparatively short inspiration. In measurements with Marey's tambour while the inspiratory part of the curve is smooth various elevations are found on the expiratory part. Respiration in the newborn infant is particularly liable to be made irregular by total interruptions due to excitement; these pauses are often strikingly long when the child cries.

It is not uncommon to find the lower part of the thorax pulled in slightly in deep inspiration (e.g., while crying); this is explained by the flexibility of the still soft thoracic wall.

(E) THE BODY TEMPERATURE

The backwardness of the functions of the newborn organism may be very clearly illustrated by the behaviour of the body temperature (Bärensprung, Förster, Sommer, Mühlmann, Eröss, Feis, Lachs, Pokrowski and others). If the temperature of a child be taken immediately after or during birth it usually shows a relatively high level, being generally between 37.0 and 38.1° C.; the temperature thus exceeds that of the mother. The height of the temperature at birth depends on the duration of pregnancy and on the development of the child; delicate and premature children usually have less high temperatures. Directly after birth the temperature falls rapidly, particularly during the first thirty to sixty minutes, and reaches its minimum after two to six hours; this may be $1\frac{1}{2}$ to 2° below the temperature recorded at birth. The temperature of the child's surroundings determines to a considerable extent the degree and duration of the fall in temperature and whether it remains at its minimum a long or a short time. The first bath usually leads to a further fall in temperature, according to Sommer on an average to 0.6° C. In spite of the numerous variations caused by the constitution and degree of development of the child, by the

surrounding temperature and by any protections against greater heat losses at the time, it is none the less permissible to consider the depression in the temperature curve as characteristic of the first day of life. When the minimum has been reached the temperature rises again, but this rise usually takes far longer than the fall. From the second day onwards it moves again within normal limits, but there is still a distinct thermolability, which may often last more than eight to ten days, i.e., until the heat regulating mechanism is functioning in as perfect a manner as is thought to be the rule in older healthy children. Eröss has shown that the temperature of a two- to six-day-old healthy infant may be markedly lowered by a lukewarm bath (27 to 30°C .) of ten minutes' duration. The customary bath of 35°C . if it lasts 2 to 3 minutes will usually depress the temperature from 37.8° to 37.0°C .

The daily variations in temperature are generally much greater in the first week than in the older infant, and may amount to $\frac{1}{2}$ to 1° or more. With regard to the daily maximum and minimum opinions differ; Eröss found the highest temperature in the morning and the lowest at noon; Lachs, on the contrary, found the highest temperature at midday and in the afternoon, and the lowest in the early morning and in the evening; this shows how much the fluctuations of the temperature must depend on the surrounding conditions. The temperature is affected not only by variations of the surrounding temperature (e.g., while the clothes are changed), but also by the intake of nutrition (suckling) and by crying; the temperature curve is usually much higher if measurements are taken an hour after lactation (Lachs); nevertheless should the temperature rise to 38° with the usual clothing and room temperature this must be considered abnormal (*vide infra*, transitory fever), in spite of the liability of the body temperature in the first days of life.

Since the general temperature level differs in individuals, sometimes being about 36° and sometimes 37° , it follows that temperature curves of newborn children show considerable variations. Average curves, such as those brought forward by Eröss, are but rarely found in individual cases; he found, apart from the highest temperature during birth, a first and a second minimum 1 to 2 hours p.p. and on the fourth to fifth day; also a first and second maximum on the second and on the sixth to eighth day. Lachs found no essential difference between the separate days (the first day of course being excepted), and his observations are probably correct.

The functional backwardness in the control of the body temperature may be due to the incomplete development of the heat-regulating centre or of the skin's regulating mechanism which in later life is capable of affecting fine adjustments such as are necessary under these conditions. There is certainly in the first place a deficient physical regulation, with an insufficiency in the factors governing heat losses. The sudden fall in the body temperature after birth may be explained in the following way (Lachs): while in the uterus the child is surrounded by amniotic fluid, immediately

after birth the evaporation of this fluid begins, the speed of this process depending on the dampness of the surrounding air. This considerable cooling must be considered mainly due to the insufficient warming of the organism by means of respiration, super-added to this the leisurely way in which the child is frequently dressed, the bath, the temperature of which is lower than that of the amniotic fluid; these factors are associated with the insufficient physical heat regulation and adequately explain the ease with which a lower temperature is reached.

Dealing merely with external influences one should take care that this cooling should not be too great. The child should be well wrapped up immediately after birth, even before the bath, and afterwards placed between hot water bottles. The bath should have a temperature of quite 35 to 40° C. and should be conducted with all possible speed, after which the child should be dressed in previously warmed clothes and napkins, well covered up or wrapped; if necessary hot water bottles should also be provided. Even though such careful procedure may be unnecessary for a strong healthy child it is nevertheless very important for delicate children, since these have a tendency toward low temperatures, and much time and care are often required before such a child can be brought to a normal temperature after a considerable fall.

(F) THE SKIN

When the child has left the vagina of the mother its body is found to be covered with a greasy, whitish layer consisting of epidermal cells and sebum (*vernix caseosa*). This should be removed as far as possible with vaseline during the first bath, which is not always an easy task, since cheesy patches are liable to remain in awkward places, especially in the ears.

The colour of the skin as it appears after the bath as soon as the child has been warmed again, is a bright red; the extremities, especially the hands and feet, frequently show a distinctly bluish coloration during the time of the temperature depression; they also feel cold. The so-called erythema neonatorum is a physiological occurrence, as is the desquamation which appears after a few days. For the sake of coherence it is not dealt with until the diseases of the skin are discussed.

The newborn head is covered more or less with hair several centimetres in length. The eyebrows and eyelashes are usually thin and very short so that they often appear to be absent. On the body exceedingly fine woolly hairs are very often found (*lanugo*). Although these are usually most plentiful in prematurely born infants they are often perfectly distinct in very well developed children, especially in the shoulder region and on the back. The most important and most interesting peculiarity which is shown by the skin of the newborn child is *icterus neonatorum*.

ICTERUS NEONATORUM.

A large number of children during the first days of life show a more or less distinct yellow coloration of the skin or well-marked jaundiced appearance. The icterus appears most frequently on the second or third day, although not uncommonly indications of it are already seen by the end of the first day. Children are never jaundiced immediately after birth; and a yellow coloration is exceptional during the first twelve hours. If, on the other hand, no trace of jaundice is present on the third day, such an appearance can hardly be expected to appear in the following days.

The yellow coloration of the skin is at first hidden by the hyperemia which is present during the first days of life, it can thus only be seen when blood is pressed out of a portion of the skin (e.g., the tip of the nose) either with the finger or with a glass slide. All parts of the body are not always uniformly coloured; sometimes the extremities, especially the palms of the hands, the soles of the feet, or the face, are still free from jaundice, while the skin of the trunk shows a distinct yellow coloration. During the following days the jaundice tends to increase. It is obvious at first sight in pronounced cases as the mixture of red and yellow gives the skin a very characteristic colour. The yellow coloration is met with not only in the outer skin, but is also quite obvious in the mucous membranes. If icterus of the conjunctiva is often unobserved, this is due to the fact that in newborn children the sclerotics are so seldom seen. As a matter of fact it is only in pronounced cases of jaundice that they are discoloured. The yellow coloration is also distinct as a rule in the buccal mucous membrane, especially in the somewhat anemic places on the gums and on the margins of the jaws.

The intensity and duration of the icterus are exceedingly variable. Usually the maximum is reached as early as the third or fourth day; it then passes off and completely disappears toward the end of the first or the beginning of the second week. Should the icterus be confined to traces it is often only visible for one to two days. Not infrequently however it lasts throughout the second week, and extends into the third and fourth weeks. Should the icterus remain visible until the second month it is an indication that a more serious primary disease is present; it does appear, however, that even "benign" icterus neonatorum may extend considerably beyond the limits of the newborn period, especially in premature infants.

The intensity of the icterus also varies within the widest limits. All degrees appear from the faintest, hardly perceptible yellow tinge to the fully developed, often even orange-yellow icterus. The general condition of the child certainly has an essential influence on the quality of the icterus. The skin of a child is often ash-coloured, but when the general condition is good and the skin well supplied with blood even the yellow colour is brighter.

The stools have no peculiarities as to colour. They are no more strongly coloured in badly jaundiced infants than in slightly or

non-jaundiced cases. The urine also does not show the usual colour of icterus urine. Even if it is possible to detect bile pigments not by the usual Gmelin's test but by more sensitive reactions (Happert, Nakajima, Obermeyer-Popper) or by chloroform extracts of the urine, the clinical fact is established that the colour of the newborn urine, especially when it has been centrifuged and filtered, is usually entirely uninfluenced by bile pigments. In rare cases, especially where undoubtedly pathological conditions are associated with icterus, dissolved bile pigments are also sometimes found in the newborn urine. Such a bilirubinuria can to a certain extent be used in differential diagnosis. This curious condition, formerly attributed to a peculiar impermeability of the kidney towards bile pigments, is explained by evidence brought forward by Parrot and Robin and also by Cruse, to show that bile pigment is present in the urinary sediment in insoluble form. It appears here in the form of golden yellow layers and granules, which are partly lying free and partly enclosed in cells or clinging to them (*masses jaunes*). Knöpfelmacher explains that the deficient solvent power of the urine for bilirubin is due to the absence or insufficiency of alkaline phosphates which are able to cause the solution of the bile pigments. In other secretions the bile pigment appears in a soluble form. This is particularly striking in the tears and conjunctival secretion in cases of conjunctivitis, these being often bright yellow in colour. The nasal secretion also sometimes shows a similarly remarkable coloration.

The liver and spleen are not enlarged in the jaundiced newborn child and the pulse rate is unaltered. We will later, when dealing with the cause of this condition, consider to what extent other symptoms, such as disturbances in the general condition, torpor, indigestion, flatulence, &c., are to be considered as symptoms of a uniform pathological condition which is the cause of *icterus neonatorum*, or merely as accidental complications or as factors in the production of the icterus.

It is difficult to make use of anatomical data as a healthy jaundiced infant has hardly ever been investigated in this connection. The internal organs of infants who have died during the time of *icterus neonatorum* usually show a distinct yellow coloration which is especially present in the internal lining of the vascular system and in the transudations and exudations. Meckel found crystals of bilirubin in the apices of the papillae of the kidney, Orth also found them in blood, brain, &c. The tissue fluid thus appears to resemble the urine in the newborn child in having a low solvent power for bile pigment. The precipitation of bilirubin in fatty tissue is explained by Knöpfelmacher as being due to the withdrawal of alkali by the fatty acids of the fat cells.

As regards the relation of absolute body weight in *icterus neonatorum*, we may consider it proved that small, especially premature children have marked jaundice more frequently and remain in that condition for a longer time than full-term infants.

If a larger number of cases be observed it will appear that icterus is somewhat more frequent in cases of low body weight than when this is higher. This is shown by the following table by Cruse:—

| Weight | Cases free from icterus |
|-----------|-------------------------|
| 1500—2750 | 10.7 per cent. |
| 2750—3000 | 11.5 " |
| 3000—3250 | 12.2 " |
| 3250—3500 | 15.9 " |
| 3500—4640 | 27.4 " |

Although these figures show that the frequency of icterus is inversely proportioned to the body weight, it cannot on the other hand be denied that very strong children with high birth weights may also be highly jaundiced.

It is stated by many writers that the losses in weight are usually greater in jaundiced infants than in the non-jaundiced. The figures obtained from Porak and Cruse show that a high degree of jaundice is correlated with a low average increase and a large average loss in weight, and with a proportionately large number of children who lose in weight during the first ten days. A comparison of the weights on the tenth to twelfth day with the weights at birth is of little value as it appears that the difference between these two numbers is to so large an extent dependent on the intake of nutrition and fluid, that the icterus in comparison can only play such a very subordinate part, that it cannot have any ætiological importance in connection with the course of the weight curve attributed to it. In this connection only the physiological loss in weight during the first three or four days comes into the question. There are certainly many jaundiced newborn children whose loss in weight is slight, while on the other hand considerable losses in weight may take place without the presence of jaundice. Moreover, even when the statistics show a correlation between *icterus neonatorum* and great loss in weight it is difficult to decide whether the children lose weight because they are jaundiced, or because they are suffering from the hypothetical primary disease associated with jaundice, or whether they become lighter and jaundiced because they have for some reason or other lost much weight. Until we can distinguish between cause and effect we must not draw any conclusions from statistics of this kind.

The statistics concerning the frequency of icterus are also very contradictory:—

| According to Sex: | in 100 per cent. |
|----------------------------|------------------|
| — " Unger | 20.2 |
| " " Quisling | 26 |
| " " Bonclon | 31 |
| " " Fuchs | 62.0 |
| " " Kehr | 75 |
| " " Porak | 78.0 |
| " " Cruse | 84.4 |
| " " Brechet in all newborn | |

The estimates are thus seen to vary enormously. The differences

are partially explicable by the probability that many slight cases of icterus are overlooked and are thus often missed out of the statistics. On the other hand it can be assumed that local hygienic conditions also take a part in so far as infectious diseases, which influence the origin or at all events the intensity of the icterus, are rare when more care is given to asepsis.

In all these statistics the most remarkable thing about all views is to the origin of the so-called icterus neonatorum is their uncertainty; the difficulty of differentiating "septic icterus" in its connection with icterus neonatorum has been referred to by many.

The subject of icterus neonatorum has already been explored from most varying standpoints for some decades and it is a favourite theme of scientific controversy. There is thus a very large number of theories regarding the nature of this icterus, and there is hardly a form of jaundice with which the icterus of the newborn has not already been enrolled. Certain differences of opinion do not concern icterus neonatorum as such so much as the pathogenesis of such forms of icterus as those with which it is brought into connection. It cannot be said today that the question of the origin of icterus neonatorum has been answered unambiguously. None the less so many important facts have been discovered, and so many observations have been made in the clinical, anatomical and histological aspects, in addition to the more or less hypothetical theories on the subject of icterus, that it is now possible to form an opinion in respect of the factors that influence the tendency of the newborn child towards icterus.

The much discussed question as to whether icterus neonatorum is to be regarded as a physiological or as a pathological occurrence is most intimately connected with its genesis. If the origin of the icterus be found in the anatomical and physiological peculiarities of the newborn child it can hardly be regarded as a disease; if it is to be attributed to the presence of infectious or other morbid influences it must naturally be regarded as a disease or as a symptom of a disease.

Whichever of these opinions is correct, this much is certain—the symptom "icterus" is not as frequent in any other period of life as it is in the first week and—a matter of particular importance—hardly ever so harmless. Whether icterus neonatorum rest on a uniform aetiological basis, or whether it be the outcome of various factors, it is none the less justifiable to speak of icterus neonatorum as a definite entity owing to the peculiar tendency shown by the newborn towards this condition. The question also arises as to why it is in the newborn child alone that this strong tendency appears.

Special efforts have been made to bring icterus neonatorum into association with anatomical peculiarities of the newborn child. The first of these attempted explanations which we must quote is the hypothesis put forward by Quincke, according to which the icterus may be traced back to the passage of bile pigments from the

intestine into the circulation. The course which this follows is said to be the ductus venosus Arantii, the foetal communication between the portal vein and inferior vena cava. Elsässer found that the ductus venosus was only closed in three cases (3·8 per cent.) out of seventy-eight infants who had died during or immediately after birth, and in twenty-three cases (hardly 12 per cent.) of 200 who died during the first days of life. In any case its lumen is very narrow. In spite of the justifiable objections which have been raised against Quinke's theory (Knopfmacher), it must be admitted that it lies by no means been disproved (Eppinger), at least in so far as concerns its essential point, which is the absorption of bile pigment from the intestine. We shall discuss this matter again later.

To Hasse we owe another explanation, attributing the icterus neonatorum to anatomical peculiarities. He considers that "the normal icterus" of the newborn is an obstructive jaundice, arising and disappearing under the influence of diaphragmatic breathing during the first days of life. The downward movement of the diaphragm during inspiration increases the pressure on and in the porta hepatis, which was already positive before the inspiration. This gives rise to pressure on the efferent ducts of the liver and on the vessels, especially the portal vein. In this way the flow of bile is chiefly hindered; it is obstructed and taken up by the body to a greater or lesser extent. The increased pressure and with it the obstruction disappear after a short time on account of the decrease in the size of the liver caused by respiration and on account of the changes in position undergone by the porta hepatis, its contained vessels and ducts, and the subjacent duodenum. The liver of the newborn is not only relatively larger and more deeply placed, but also exhibits a peculiar condition of the under surface, especially of the quadrate and Spiegelian lobes, which form the boundaries of the porta hepatis. About half of the Spiegelian lobe is placed on the under surface of the liver, but it projects so far forward that the floor of the hilus is very deeply situated and bent backwards. The quadrate lobe has a hooked process directed posteriorly in front of the porta hepatis, subsequently passing over the floor of this. The first part of the duodenum has an anterior flexure, and consequently runs posteriorly and downwards. As a result of this arrangement the ducts of the liver and the vessels are wedged in between the two lobes of the liver and the first part of the duodenum. It thus occurs that the blood-pressure in the large portal vein, which before birth is fed by the umbilical vein, and in which the blood flow is stopped after birth by each inspiration, retards and hinders the flow of bile in the adjacent bile duct. When breathing commences the downward movement of the diaphragm, besides raising the pressure in the portal vein, presses the liver against the anterior abdominal wall and the subjacent intestines, and in this way increases the pressure bearing on the bile duct. The quadrate lobe is hollowed out by the stomach

and pressed downwards and forwards, while the Spigelian lobe is pushed back upwards and comes to lie perpendicularly. As the liver decreases in size its under surface is raised. This results in an enlargement, flattening and extension of the porta hepatis, and a consequent facilitation of the flow of bile.

According to this description *icterus neonatorum* is a mechanical *icterus*. While Hasse attributes it to the anatomical peculiarities of the liver and assumes a pressure which leads to obstruction of the large bile ducts at the porta hepatis, others seek the origin of the obstruction in the interior of the liver.

Cruse supposes a desquamative catarrh of the bile passages to be the cause of the biliary obstruction; Kehrer suggests congenital stenosis of the biliary system; Birch-Hirschfeld an oedema of Glisson's capsule. None of these opinions receive anatomical support. A number of writers (Weber, Wernel, Silbermann) suggest that the capillaries of the liver, being overfilled and distended with blood, press the sides of the biliary passages together. This theory could not be verified by the histological investigations of Abramow and Knopfmacher, both of whom found the bile capillaries gaping instead of compressed. It thus appears that it is not these but more peripherally placed biliary passages which are compressed.

Virchow thought that the origin of the obstruction was to be sought in the stoppage of the bile due to a mucous plug due to catarrhal inflammation. He concluded that the changes were those usually considered to be the causes of *icterus catarrhalis*, but the clinical symptoms of *icterus neonatorum*, especially the fact that bile is not absent from the stools, seem to contradict this analogy. Nevertheless, similar ideas are continually cropping up even at the present time. Opitz, for instance, holds that *icterus neonatorum* should be regarded as a mild form of catarrhal jaundice. In his opinion it is a case of slight swelling of the mucous membrane which partially obstructs the passage of the bile during the first days, at a time when it is flowing in particularly large amount. Opitz maintains that plentiful nourishment during the first four days predisposes towards the onset of *icterus*. He found a striking difference between boys and girls; in his cases 44·3 per cent. of boys became jaundiced as against 33·7 per cent. of girls. Although his very comprehensive statistics show that on the average girls drink more than boys, Opitz thinks that an addition of 70 gm. of milk to the diet will cause *icterus* in the boy who is already strongly predisposed towards that condition, while with girls 200 gm. are necessary. Opitz also assumes another peculiar disposition to *icterus* apart from the catarrhal swelling of the duodenal and biliary mucous membrane, namely, an exceptional sensitiveness of the mucous membrane of the boy, as also of the weakly and the artificially fed child.

The assumption that the catarrh of the mucous membranes is due to excessive feeding appears not to be quite correct, inasmuch

as the icterus is usually present on the second day at a time when the quantity of drink is, with few exceptions, still very small; it also tends to diminish towards the end of the first week while the amount of drink is increasing. It is, moreover, probably correct to assume that the intestinal irritation during the first days of life primarily affects the large intestine and does not attack the upper part of the small intestine. Even if the opinion be accepted that a duodenal catarrh plays a part in the genesis of icterus neonatorum, it remains unexplained why a similar catarrhal icterus practically never appears in later infancy, in spite of frequent and much more violent intestinal catarrhs.

The histological investigations of Abramow and Knöpfelmacher tell against all attempts to explain icterus neonatorum as a mechanical icterus in the usual sense; they were unable to find the characteristic evidence for obstructive jaundice in the livers of jaundiced infants. Both observers made use of Eppinger's method, which permits of an exact representation of the bile capillaries. As with Eppinger, who himself investigated a few cases of icterus neonatorum, they were not able, in a single case, to observe the diagnostic sign of obstructive icterus—namely, rupture of the bile capillaries in the livers of newborn children. The bile passages were shown to be unobstructed and their walls intact. No apparent causes of obstruction could be seen, nor any consequences of such a condition which could support the view that a migration of obstructed bile had taken place into the surrounding blood-vessels.

It thus appears that during the first two or three days of life an abnormal filling of the bile vessels and a varicose enlargement of the bile capillaries may be observed, and that this widening of the bile ducts first takes place after birth. Knöpfelmacher brings this histological condition into relation with the results of determinations of the bile-viscosity among newborn children. At the time of birth the viscosity is relatively high; the bile capillaries are therefore filled with somewhat more tenacious bile at this time, the forward movement of which demands a higher secretion pressure. The increased secretion of bile which takes place immediately after birth increases the demand on the secretion pressure. The mechanical work necessary to drive the richly flowing secretion out of the ducts filled with viscous bile is too great for the liver cells of the newborn child to perform; an obstruction results and the passages dilate. Were ruptures to be found in the dilated bile capillaries, icterus neonatorum could be explained satisfactorily as a mechanical icterus. This is not the case, however, and the opinion is forced upon one that the passage of bile into the blood takes place in another way. The constitution of the liver cells of the newborn must be taken into account.

Abramow supposes that there is a lowering of the energy of excretion, which he thinks is caused by their being unused to work, and an impairment of function of the cells similar to that occurring in infectious diseases (asthenic hypercholia). As in the latter

the cell injury, so in the newborn the impairment of the cell function is shown by a diminished energy of secretion and by a probable increase in the productive power of the cell, since it results in the hypercholelia due to hyperamnia. In this view the essential factor in the occurrence of icterus neonatorum is a loss of equilibrium between the power of excretion and secretion.

The bile reaches the blood, according to Knöpfelmacher's view, by direct passage from the liver cell itself into the blood or lymph. A similar phenomenon may occur, as has been described in the functional disturbances of the liver by Minkowski as "*Icterus per diapedesin*," by E. Pick as "*Paracholie*." In support of his view that an absorption of bile in the liver is possible in the absence of any obstruction, Knöpfelmacher lays stress upon the excretion of bile pigments in the urine of starved animals. Gundobin also attributes icterus neonatorum to an absorption of bile into the blood and lymph as a result of secretory anomalies. He does not, however, agree that an increased formation of bile takes place, but attributes the anomalies of secretion to disturbances in the cell nutrition due to obstruction of the circulation of the blood.

If the view be correct that the occurrence of icterus neonatorum is finally due to an insufficient function of the liver cells, a further question arises as to whether this insufficiency in the function of the cells be due to backwardness of the infantile organism alone or to cellular injury. Both views receive support. In favour of the cellular backwardness is the relative frequency and intensity of icterus in prematurely born children; in favour of injury to the cells is the coincidence of icterus with symptoms of disease, which has been described by many authorities.

An injury to the cells may be caused by infectious disease or by toxic substances. As Pick has said, it may be a case of paracholia due to infection (*Infections-toxicationsparacholia*) or of paracholia due to auto-intoxication (*auto-intoxicationsparacholia*). Although umbilical sepsis is often associated with icterus, it is only in rare cases that umbilical infection can be made responsible for typical icterus of the newborn child. According to Czerny-Keller, icterus neonatorum is caused by infection from the intestine. Czerny founds his view on his experiences in maternity clinics and foundling institutions which showed that icterus neonatorum disappears at times and periodically becomes more frequent; that it sometimes appears in a severe and then in a mild form, and that it is particularly frequent when possibilities of infection are very plentiful. This view is also supported by Unger. In a statistical study of the cases of icterus in a maternity clinic he found that the frequency showed great variations in the different months; this he attributes in the first place to hygienic factors. It appeared that the increase and decrease in the icterus cases were dependent upon the regular disinfection and cleaning of the various clinical departments. It happened repeatedly that in clean wards no cases of icterus were observed for weeks at a time, while the cases became

more frequent the longer the time since the last cleaning. Czerny also considers icterus graecus to be not essentially different from icterus neonatorum, which in the great majority of cases pursues a favourable course, and sees in these two conditions only the type of a single group of diseases which includes all transitional forms from one extreme to the other. He explains the ready occurrence of icterus neonatorum by the permeability of the intestinal wall for bacteria which is present in the first week of life. The statement of Czerny, that in the lying-in room icterus neonatorum appears at the same time as puerperal infection, implies that it is due to infection with pathogenic bacteria. The icterus itself may therefore be among the symptoms of an intestinal septic infection, even if the latter be very slight.

It is very probable that external circumstances exercise an influence on the occurrence and course of icterus neonatorum, as is shown by the different statements as to the frequency of its occurrence and the variations in the time of its appearance. Nevertheless, it appears very questionable whether the most careful puerperal nursing would be able to prevent entirely the occurrence of icterus neonatorum. One must assume that such infections occur exceedingly easily—so easily that they can no more be absolutely avoided than can the settling of the normal bacteria in the buccal cavity and in the intestine. It is, moreover, not improbable that this "physiological" infection of the alimentary canal may itself take a part in the genesis of icterus neonatorum, as under certain circumstances intestinal toxins may make their way through the relatively easily permeable intestinal wall of the newborn and damage the function of the relatively easily affected liver cells.

Although it must be admitted from the above that a connection exists between processes in the intestine and icterus neonatorum, a paramount importance of intestinal infection as a cause is made less likely by the fact that the icterus usually tends to be visible as soon as twenty-four hours after birth, or even sooner, at a time when the infection of the contents of the intestine has only just begun; such an infection must then be attributed to the swallowing of vaginal secretions containing bacteria during birth, a mode of infection on which the hygiene of the period of the puerperium naturally has no influence. Since, moreover, icterus occurs only very exceptionally in older infants who are ill in spite of the high permeability of the intestinal wall also existing in them, there must in any case be other causes which make possible the easy occurrence of icterus in the newborn.

Besides the mechanical forces and the functions of the liver cells a third factor in the genesis of icterus neonatorum must be considered—namely, the quantitative and qualitative peculiarities of the bile itself. As has already been stated, Abramow deduces from the disproportion he found between the abilities of excretion and secretion of the liver, an increased power of production which (on account of the hyperæmia arising after birth) leads to a condition of hypercholelia.

itself which is followed by the passage of pigment out of the erythrocytes. It is possibly unjustifiable to disregard the fact that in the newborn child especially more or less copious hæmorrhage takes place into the skin, the mucous membranes and the internal organs, on account of the congestion taking place during birth which varies with the duration of labour and the degree of asphyxia. In this connection it is noteworthy that icterus seems to be distinctly more frequent after long and difficult labour than after births of short duration (Wermel and others). Here it is certainly possible for a hyperæmia from congestion alone to favour the occurrence of icterus without its being necessary for hæmorrhage to play a part. Nevertheless the causal connection between hæmorrhage and icterus is supported in a convincing way by the observation that in cases with severe internal hæmorrhage the icterus sometimes tends to be remarkably intense (Raudnitz, Skormin). The acceptance of an icterus pleiochromicus is certainly permissible in such cases.

In an otherwise healthy and strong infant the author observed a hæmorrhagic disorder (considerable extravasation in the region of the caput succedaneum, epistaxis, intestinal hæmorrhage, decided fall in the coagulability of the blood, in the course of which an exceptionally intense icterus developed with dark orange-yellow colour and decided bilirubinuria). The hæmorrhages ended at the end of the first week and the icterus disappeared during the second (see fig. 82).

Although such cases also afford very convincing evidence for a causal connection between icterus and hæmorrhage it cannot be denied that internal hæmorrhages take place in which the icterus is not of a very high degree. At all events it cannot be maintained that a direct parallel exists between hæmorrhage and icterus, and that icterus neonatorum is dependent on the intensity of the hæmorrhage alone. Such an opinion would be *a priori* untenable, moreover, on account of our experiences in the pathology of later life; thus purpura is by no means frequently associated with particularly marked icterus.

It has not been convincingly proved that on account of the destruction of corpuscles from the placental blood, particularly frequent and severe icterus is shown by children whose umbilical cords are ligatured late and who thus contain a larger amount of such reserve blood (Hofmeier, Affanasiew and others).

If hæmorrhages be concerned in the production of icterus neonatorum this can only be the case in so far as the material for an increase in the pigment and thus in the secretion of bile, is provided in this way. Whether or not it results in the occurrence of icterus depends on other factors.

The destruction of blood corpuscles during the first days of life may be looked on as a direct consequence of the excessive number of corpuscles present after birth; that is to say, as an adjustment of the number of blood corpuscles to a normal level. On the other hand it may be supposed that some sort of toxic influence leads to

a destruction of corpuscles, or that as Heimann imagines, blood proteins are attacked when the nourishment taken is insufficiently assimilated. To decide this question the results of daily counts of the erythrocytes have most frequently been brought forward. In this connection it must be pointed out that the thickening of the blood which takes place during the first days on account of the physiological loss of water (exactly at the time when the occurrence of icterus is determined), makes the determination of results very difficult. The figures derived from daily blood counts do not at any rate afford convincing evidence for a destruction of erythrocytes as being related to the appearance of icterus.

In jaundiced infants Heimann found a rise in the number of red corpuscles from the third to the fourth day, followed by a steady fall; in non-jaundiced the fall had already begun on the third day. Further in jaundiced children a fall in white corpuscles takes place from the third to the fourth day, and then a slow rise, whereas in the non-jaundiced there is a continuous fall from the third day. Jaundiced and non-jaundiced children already show approximately equal values by the sixth day. The proportions of the different forms of leucocytes show approximately equal relationships in the two cases, only the eosinophils are a little more frequent in cases of icterus.

Knopfmacher is of opinion that the number of red corpuscles during the first week of life is independent of the developing icterus and that its variations are to be attributed merely to the changes in the amount of plasma.

According to the investigations of Cohnstein and Zuntz the number of blood corpuscles in the newborn animal does not diminish during the first days of life, but rises steadily.

In considering the results of blood counts it must at all events be borne in mind that an increased destruction of erythrocytes is not necessarily followed by a fall in their number. The disturbance may be compensated by an increased production. The fact that during the first days of life a new formation of red blood cells takes place is shown by the presence of nucleated elements. It is quite possible that at the time of birth an increase in the metabolism of the blood takes place, so that a considerable number of erythrocytes are destroyed giving rise to pleiochromia, but that at the same time the regenerative functions of the blood-forming organs are stimulated so that the number of blood corpuscles in the circulating blood is greatly influenced.

Leuret has found free haemoglobin in the blood of jaundiced newborn children, discoveries which, however, are quite isolated. On careful examination, at all events, the serum of the newborn shows no signs of haemoglobinemia.

Since a lowering of the resistance of the red corpuscles is found in the so-called haemolytic icterus, results on this point are not without interest concerning the question of icterus neonatorum in the newly born. At the moment of birth the blood of the infant

and also of the umbilical cord has the same resistance as that of the adult. Opinions differ as to what occurs on the following days. Whereas Knöpfelmacher could not find any alteration whatever during the first week, even in cases of marked icterus, the resistance, according to Slingenberg, of the red corpuscles increases in all infants and is greatest, on an average, on the third or fourth day; it then diminishes again and usually reaches a constant level about the tenth day. The increase in resistance is most marked in jaundiced children. Slingenberg points out that in this connection there is an analogy with the alteration in resistance which is observed in icterus due to obstruction, and he attributes the considerable increase of resistance in jaundiced infants to the passage of bile into the blood. Therefore the diminished resistance, especially associated with hemolytic icterus, does not occur in icterus neonatorum. Similar results were obtained by Unger and Graff; the resistive power of the red corpuscles rises simultaneously with the icterus and remains throughout its duration. The resistance certainly rises also in non-jaundiced infants but this rise is not nearly so great as in the jaundiced child.

The results of microscopic investigation show that hemoglobin passes out from the corpuscles. Silbermann and Scherenziss found an increased number of ghosts in the blood of the newborn and deduce from this that the hemoglobin is less firmly combined with the red corpuscles of the newborn child. It still remains uncertain whether the hemoglobin freed in this way is of importance in the production of icterus.

If from what has been said, it is still doubtful whether the bile of the newborn be relatively rich in pigments or whether besides the polycholia a porriochromia is also present, none the less it can be taken as proved that the absolute amount of pigment formed in the liver and passed into the intestine is a comparatively considerable one.

As already mentioned Quinke attributes icterus neonatorum to the fact that bile pigments present in the contents of the intestine are partially absorbed and taken up into the general circulation. Quinke supposed that the pigment was derived from the meconium. To disprove this view it is hardly necessary to refer to the fact that a premature evacuation of the meconium by natural or artificial means is unable to prevent the occurrence of icterus (Kehrer, Knöpfelmacher), as it is sufficient to note that icterus neonatorum may last three weeks and thus extends into a period when the effects of the meconium can hardly be a determining factor. It is not justifiable to attribute the increase in pigment merely to the meconium. Owing to polycholia the stools of the lactation period, excreted after the meconium period, are also usually very deeply pigmented and obviously contain an excess of bile pigment (Mensi). By means of a duodenal catheter A. F. Hess has demonstrated the presence of a large amount of bile pigment in the

upper parts of the intestine. It is possible that part of this pigment is reabsorbed, a process in which the permeability of the intestine during the first days may play a part. Should this opinion be correct and the bile pigments really flow back to the liver through the portal vein, the assumption of a patent ductus venosus is quite unnecessary. It is possible that the conditions present are similar to those of the so-called urobilin icterus. For this condition the following explanation is generally accepted: From the reduction processes in the intestine a pigment is formed which is either identical with urobilin or chemically similar; this is partially absorbed and taken back in the liver, there to be reconverted into bilirubin. Should the liver cells be absolutely or relatively insufficient, i.e., injured in function or not able to cope with an increased amount of pigment, they will no longer take up the urobilin so that the substance enters the general circulation and appears in the urine. In the newborn child urobilinuria does not usually develop as intestinal reduction processes tend to be absent, and therefore no urobilin is formed in the intestine. But should this happen exceptionally, the author's observations go to prove that urobilin is very liable to be passed in the urine of the newborn child. The yellow coloration of the skin accompanying urobilinuria is derived also in urobilin icterus from the bilirubin and not from the urobilin. To put the case more simply, it appears that in icterus neonatorum the bilirubin passing up from the intestine is not adequately dealt with by the liver cells, which are relatively insufficient, backward, injured or over-burdened with pigment; the bilirubin therefore passes into the general circulation and saturates the tissues.

Possibly some renal obstruction of the bile pigment is also present. The urine is certainly not free from bilirubin, but contains relatively small amounts of that pigment. Accurate observations concerning a low permeability of the newborn kidney have at all events not yet been made. The methylene-blue researches of Reusing and Lereboullet are not conclusive and could not be confirmed by Laurent. The increased concentration of the blood and the low secretion of urine at the time of the physiological loss of weight might be brought forward as accessory factors in the theory of a pigment obstruction in the blood. According to Biffi and Galli the blood plasma is richer in bile pigment during the first week than at other times.

If one surveys and criticizes closely the many different views concerning the causes of icterus neonatorum, one is driven to the conclusion that it can hardly be correct to give one single cause for icterus neonatorum. It might be possible to reconcile the conflicting theories of those who see in icterus neonatorum a physiological peculiarity of the newborn child, and those who look on it as a disease, if instead of the controversial subject of "icterus neonatorum" one attempted to explain the "tendency of the newborn to icterus." Before an attempt is made to deal with the question

as to whether icterus is caused by physiological or pathological factors, it is necessary to answer another—why does the newborn child so easily and so frequently become jaundiced? In explanation of this tendency the following points may be brought forward:—

1. Certain mechanical factors, which are partly of anatomical origin and which may partly be due to the qualities of the bile itself;

2. The richness in pigments of the newborn organism, which is shown either by pleiochromia of the bile or by a polycholia and due to a high production of pigment (possibly partially existing in intra-uterine life) and possibly also connected with congestion and hæmorrhages arising during parturition.¹

3. The condition of the liver cells, which on the one hand may show a congenital (physiological) functional backwardness and on the other hand are very easily injured by toxic and infective influences. The permeability of the intestinal wall of the newborn offers ample facilities for the action of the latter.

Evidently the equilibrium between cell function and the amount of conveyed pigment can very easily be disturbed. The icterus may occur under certain conditions, which may be regarded as physiological. Should definite pathological influences arise these may result all the more easily in the occurrence of icterus or may influence its intensity and duration.

THE MAMMARY GLANDS OF THE INFANT.

Just after birth the mammary gland of the infant is represented by a nodule, the average size being equal to that of a hemp seed, sometimes smaller but rarely larger. In the course of the first three to four days it enlarges and usually reaches the size of a pea. If at this time the gland be pressed lightly, there will flow from the centre of the nipple, which is usually depressed, a drop of thin fluid, at first colourless, later milky and cloudy; when examined microscopically this is seen to consist of the same elements as the milk of the nursing woman, namely, fat droplets and more or less

¹ NORDSTRÖM, CONNECTION OF ICTERUS.—Important discoveries have recently been made by Ada Hirsch (*Ges. f. intern. Med. u. Kinderheilk.* in Wien, July 3, 1913). By means of the colimetric method of Hyman v. d. Bergh and Snapper, she was able to show that the serum of the blood of the umbilical cord always showed very large quantities of bilirubin, and that the higher the amount of the bilirubin content of the blood from the umbilical cord the quicker was the onset and the more marked the progress of the subsequent icterus. These conditions show that all factors which only influence the child after birth cannot be regarded as primary causes of icterus neonatorum. Further researches showed that in all children the bilirubin content of the serum rose rapidly during the first days. The organism appears to contain plentiful amounts of bile pigment even at the moment of birth; the discharge of this substance into the blood, however, takes place chiefly only after birth and may well be connected with the beginning of the vital processes of extra-uterine life. Whether the tissues inhibit bile pigment, i.e., whether icterus occurs, depends on whether the bilirubin value of the serum exceeds a certain level.

numerous colostrum cells. With regard to its chemical composition, the secretion of the infantile breast most closely resembles the colostrum of the woman before delivery. In a fourteen-days-old child (with considerable swelling of the mammary glands) Genser found that it had the following composition:—

| | | |
|--------------------|-----|--------|
| Water | — | 95.701 |
| Solid constituents | — | 4.299 |
| Casein | — | 0.557 |
| Albumin | — | 0.499 |
| Fat | ... | 1.436 |
| Lactose | ... | 0.056 |
| Salts | — | 0.246 |

The time at which secretion can be pressed out of the mammary gland of the infant is usually somewhat later than the time at which the secretion of milk begins in the breast of the mother. Often five or six days elapse before the secretion is apparent, nevertheless there are plenty of cases in which secretion may easily be pressed out as early as the fourth day. A still earlier occurrence of the condition, about the first or second day, is an exceptional event; in such cases the secretion is usually watery. According to Siney histological investigations have shown the beginnings of the process of secretion even during foetal life, this reaches its greatest height between the fourth and tenth day after birth. The swelling and secretion increase until the beginning or middle of the second week, though the intensity varies greatly in different individuals. Sometimes the glands remain the size of a pea or of a hemp seed, in other cases they swell, forming tumours as big as a hazelnut, cherry or walnut. The mammary glands then appear like veritable small mammae, which show a firm consistency like those of the woman at the time of commencing lactation or in a condition of engorgement. If the glands be pressed at the time the secretion sometimes spurts out. Even in very marked cases the glands tend to diminish after two to three weeks. If secretion and swelling are moderate these may have entirely disappeared after three to four weeks, nevertheless milk may very frequently be found in the child's breast in the second month, not infrequently also in the third and in exceptional cases even in the fifth month (Basch). Runge even maintains that in many cases (?) a small amount of secretion may be found until the end of the second year.

In the full-time infant the milk secretion is very rarely absent, in the premature child its non-appearance is far more frequent. In cases in which the parenchyma of the gland is less developed—this appearing especially the case in small premature infants—the enlargement of the organ and the secretion arising after birth are small or entirely absent. It must, however, be assumed that in the full-time child the presence of parenchyma capable of secretion in both sexes is the rule, and therefore the excretion of milk must also be regarded as a physiological occurrence.

Formerly the secretory process of the infantile breast was

attributed to the increasing concentration of the blood after birth and attempts were made to explain it as being due to a fatty degeneration of the gland cells or to a process of desquamation. To-day the phenomenon is regarded generally as a true glandular function, which ceases in course of time just as the secretion of the maternal gland when this is not evacuated. If the question of the origin of the hyperplasia and secretion of the glands be considered it will be seen that the opinion of Knöpfelmacher is most probable that the same factors are at work as in the case of the maternal glands. According to Basch, the necessary hormones are supplied to the foetus by the mother simply through the circulation of the blood and are probably not to be regarded as substances produced by the foetus; it is much more probable that they are the same as the hormones of the ovary and placenta. In animals Basch was able to show their presence in a watery extract of the foetus.

If a comparison is made between the intensity of function of the infantile glands and the yield of the maternal breast, in the opinion of Basch, a parallelism may generally be shown, in so far that when the infant's breast secretes plentifully that of the mother tends to produce a large amount of milk. The reverse does not hold good since a small development and deficient secretion of the infant's breast does not imply a similar condition on the part of the maternal glands; a marked disproportion is often seen in this respect.

A "treatment" of the secreting mammary glands of the child is entirely superfluous. So far as possible they should be left alone and not pressed too much. If considerable symptoms of engorgement are present, it is probable that these are as unpleasant to the child as is the corresponding condition to the mother; in these cases the therapeutic procedure is always the same as when signs of obstruction are present in the breasts of a nursing woman: poulticing with aluminium acetate and application of a light bandage. The pumping off of the secretion is useless because it is undesirable to maintain the secretion and when pumping ceases fresh symptoms of engorgement would appear.

The well-known custom amongst the poor of squeezing out the "witch's milk," must be looked on as a bad habit. If this takes place, as must often be the case, with dirty hands an infection may easily take place, possibly also the injury caused by repeated and careless squeezing helps to produce inflammation. Micro-organisms are found in the secretory ducts of the infantile breast just as they are in the maternal, and it can be understood that any of these may give rise to symptoms of inflammation in an engorged breast; injuries may be accessory factors in giving rise to the condition.

The occurrence of mastitis is shown by a considerable swelling of a gland, oedema, redness and rise in temperature of the skin covering it. The beginning of the disease usually occurs in the second or third week. Since the mastitis is almost always

unilateral, any great difference in size between the two breasts is an important diagnostic sign. Beginners often mistake the considerable swelling due to engorgement for mastitis. As in the woman so also in the child the inflamed parts of the gland may form an abscess which discharges spontaneously. In rare cases the inflammatory process also affects the connective tissue surrounding the gland; a perimastitis results, which may lead to considerable infiltration sometimes as far as the axilla (Runge). Such cases may lead to general infection, a condition which only occurs in quite exceptional cases with simple mastitis in the newborn.

Should symptoms of incipient mastitis be present, it may be possible to hinder its progress by frequent application of a small Bier's cup. The evacuation of the excretion by this means is certainly practicable. Otherwise attempts should be made to combat the inflammation by poulticing with a weak solution of aluminium acetate, 50 per cent. alcohol, &c. If fluctuation be observed the pus must be removed by means of a small, radiating, and if possible, peripheral incision, the wound to be treated according to general surgical principles. The prognosis is favourable.

The literature contains records of several cases of agnathous congenital hypertrophy of the mammary gland in girls, which may be combined with premature sexual development; such cases are rare (Jubler).

(G) NERVES AND ORGANS OF SENSE. PSYCHIC CONDITIONS

Experiments on the excitability of the cerebral cortex of newborn animals (Soltmann and others) have shown that in some animals, during the first week, stimulation of the cortex produces little or no muscular contraction. The results of these animal experiments cannot be applied to man, because of the clinical fact that, in cases of subdural hæmatomata, cortical convulsions occur in the very first days; these are hardly to be distinguished from those occurring in older infants and thus prove the possibility of a stimulation of the muscles passing out from the cerebral cortex. This by no means makes it impossible for the motor centre of the cortex to be imperfectly developed in the first period of life. Gandobin considers above all that there is an insufficient development of the inhibitory centres of the brain, and in this way he seeks to explain the increase in the reflexes occurring in infants of the first week, also the slight flexor contractures of the extremities observed in the majority of newborn infants.

An anatomical foundation for the backwardness of the development of the nervous system exists in the imperfect myelination in the region of the pyramidal tracts, the incomplete development of the peripheral mixed and sensory nerves and also of the nerves connected with the organs of sense; a physiological foundation is shown by the behaviour of the peripheral nerves towards the electric

current. The galvanic excitability is relatively small during the first week of life; by electrical investigation reactions are elicited which resemble the reaction of degeneration. The excitability of muscle by the faradic current is also relatively low both on direct and indirect stimulation. Westphal found that children under three weeks old remained comparatively quiet when stimulated by currents so strong as to be almost unendurable by adults; it is difficult to decide how far this is due to incomplete development of the peripheral nerve tracts or to backwardness of the central pain receptors.

Opinions concerning the condition of the various reflexes in the newborn are by no means in agreement. This fact is doubtless due in part to the fact that some reflexes, even when they are quite active, cannot always be elicited owing to the low tone of the muscles of the extremities.

The knee-jerk can almost always be elicited as early as the very first day and is usually fairly active. During the first weeks Furmann was frequently able to obtain reflexes on both sides of various strengths.

According to Furmann the ankle-jerk is positive in 60 per cent. of cases. Bychowski was only able to elicit it four times out of sixty-four children under six months old.

Babinski's plantar reflex is, according to Furmann, positive in the majority of cases. Stroking of the inner border of the sole always produces dorsiflexion of the big toe under normal conditions (Engstler). The reflexes are often very brisk.

According to Furmann the cremaster reflex is positive in the majority of cases in the first six weeks; Cattaneo was not able to observe it during the first three months; Peritz thinks it is seldom present; Faragó, on the other hand, only failed to obtain it in 10 per cent. of cases.

Furmann considers that the abdominal reflex is but seldom observed in the first period of life; Bychowski also does not count it among the congenital reflexes; Faragó, on the other hand, was always successful in eliciting it (in 117 children) by stroking the region above the mons veneris with a needle.

The pharyngeal and nasal reflexes are usually but not always observed, premature infants especially often show a very slight reaction in this respect.

Moro calls attention to the fact that when the typical point is percussed in infants of the first week they react with a true facial phenomenon, in which the twitching is not always confined to the corresponding facial region, a contraction occurring sometimes on the side which is not percussed. The same applies to the mouth phenomenon of Escherich or the lip phenomenon of Thiemisch (twitching of the orbicularis oris on percussing the upper lip or the angle of the mouth) and to the eyelid reflex (contraction of the orbicularis oculi on tapping the glabella and its neighbourhood). According to Moro all the facial phenomena mentioned are to be

regarded as facial reflexes and merely the expression of the heightened excitability of the reflexes during the first period of life. The "Fressreflex" of Oppenheim (rhythmic swallowing on stroking the lips) is also found in young infants under normal circumstances.

ORGANS OF SENSE.

Eye.—It may be concluded from the numerous observations at hand (Kussmaul, Preyer, Roehmann and Witkowski, &c.) that the newborn child is sensitive to light, being able to distinguish between light and dark, either directly after birth, or at any rate a few hours later. True sight or fixation is, however, not present, either in the newborn or even throughout the first weeks of life. Even though it may happen that children during the first fortnight do turn their heads towards a source of light this is due, according to Preyer, merely to the pleasant subjective feeling produced by it, and does not involve the recognition of the object apparently looked for. Corresponding to the absence of conscious vision, the eyes of the newborn child, both when asleep and when awake, not infrequently perform very inco-ordinated movements; one eye moves to the right, while at the same time the other moves to the left. The movements take place chiefly in a horizontal plane; nevertheless, vertical movements of the eyeball also occur together with decided deviations in height between the two sides. Very often strong movements of convergence may be observed, especially during the first two weeks of life; some children still squint in the second month, such strabismus not necessarily being a pathological symptom. Even though it may happen that children in many cases move the eyes together as early as the first day of life, or at least appear to do so, they are, in reality, unconscious movements which have nothing to do with real sight.

The newborn child usually keeps its eyes closed. When one eye is opened the other often remains closed, thereby causing a peculiar asymmetry of the movements of the eyelid; the eyes are frequently opened to a different extent, and eyes and lids often move quite independently of each other.

The light reflex of the pupil is congenital. Furmann made the observation that a sudden light will cause contraction of the pupil of the newborn, but the narrowed pupil again dilates, sometimes after no more than two to three seconds. This subsequent enlargement of the pupil is frequently found in premature infants, evidently a consequence of the imperfect development of the nerve tracts and of the exhaustion of their energy. Since no focusing takes place the convergence reflex of the pupil is usually absent. Preyer saw, nevertheless, that during the first two to six weeks of life, by bringing a candle flame or a bright object towards the face of a young infant, a convergence of the visual lines may take place; by observation of the lens image it was discovered that a contraction of the

muscles of accommodation is associated with it. "Narrowing of the pupil, increased thickness of the lens, and convergence occur together when the infant is approached by a light without its being permissible to infer the slightest volition."

The considerable narrowing of the pupil during sleep is absent in the newborn and in the first weeks; this is doubtless associated with the still incomplete myelination of the oculomotor and optic nerves, especially the latter (Gudden).

The action of strong light is followed by reflex closing of the eyelids as early as the first days. Children often shrink when approached by a strong source of light. But the symptom of fright is absent, with the twitching of the eyes (psychoreflex). One might pretend to strike at the face of the infant without it taking the slightest notice—only contact will cause the eyelids to close. The corneal reflex is also present; though Preyer observed that during the first three months sprinkling of the eyes during the bath does not make the eyelids close.

It is impossible, therefore, to form any opinion as to the faculty of vision of the newborn child from the above reactions to visual impressions and from the observation of the eyes.

It is a widespread custom to cover the face of a newborn child with a cloth in order to shade the eyes from the injurious effects of light. Since the reflex closing of the eyes in a strong light is present already at birth such fears are without much foundation. A certain photophobia appears to exist at first; children turn their faces towards the light only after a few days, while before then they appear to find strong light unpleasant. A newborn child should not be placed in direct sunlight or in the glare of a bright lamp, but in a more shaded part of the room; the covering of the face and eyes is not only quite superfluous but even injurious, as fresh air is kept from the child.

The outwardly visible anatomical peculiarities of the eye of the newborn are the greater thickness of the cornea, the shallowness of the anterior chamber of the eye, and the absence of pigment from the iris. The last-named is the cause of the blue eyes of the young infant. As a matter of fact it is not a definite blue; the iris has usually a grayish violet appearance. Brown coloration of the iris is practically never seen, even in strongly pigmented black-haired children.

Organ of Hearing.—All children are deaf immediately after birth, at least for noises and sounds which are not high-pitched or particularly loud. This is due to the fact that before breathing takes place there is no air in the tympanic cavity. It is not until breathing has taken place for several hours that air appears among the yellow fluid and gelatinous tissue that fill the tympanic cavity of the newborn; a few respirations are not sufficient to effect this filling with air (Lesser). Besides the absence of air in the tympanic cavity another cause of deafness in the newborn must be considered, viz., the closure of the outer ear owing to the approximation of its

sides. According to the time taken before the tubes and passages of the auditory apparatus are open the reaction to sound appears after different periods of time in different children. Most children react with reflex contraction of the muscles of the face as early as the first days of life to loud and especially to high sounds. Kuvirt was able to show that a large number of infants reacted to the sound of a tuning-fork within the first twenty-four hours; Alexander also believes that the child possesses a positive and not inconsiderable auditory sense as early as a few days after birth. The turning of the head towards the direction of sound does not take place before the second week, usually still later.

The reaction towards acoustic impressions differs greatly in individuals, at all events it is not permissible to give a definite decision with regard to the power of hearing during the newborn period. According to Preyer, deaf mutism may be suspected when a full-time child after the fourth week shows no movement when a loud noise is made behind it.

The reflex nystagmus caused by the labyrinth belongs phylogenetically to the oldest reflexes. Alexander, from experience of large number of cases, found in 78 per cent. of cases in the newborn a reflex excitability, immediate occurrence of nystagmus on stopping the rotation of a revolving chair; in a number of cases, after stopping the rotation movements of the eyeball took place at first without any detectable nystagmus, which did not occur till later. Twenty-two per cent. of full-time newborn children did not react normally; but the abnormal reflex excitability generally appears to change to normal within the first week of life. Premature infants exhibit a similar condition.

The Sense of Taste.—The newborn child has not only a sense of taste in general, but is also able to distinguish between the qualities of taste (sweet, sour, salt and bitter). Kussmaul by tests with cane sugar, common salt, tartaric acid and quinine, has proved that the reactions differ according to the different substances. Sweet substances evoke an expression of pleasure and produce voracious sucking movements, while bitter, sour and salt substances, if the solution is not too much diluted, cause distortion of the face, movements of retching and crying. Individual differences are very great also on this point. Preyer's observations on an anencephalus show that all taste reflexes take place without the participation of the cerebrum.

The Sense of Smell.—That the newborn child possesses sense of smell is shown by the fact that it refuses to drink when small amounts of substances having an unpleasant smell are placed on the mother's breast or on the teat of the bottle; the power of smell seems to be already present on the first day of life (Preyer).

The Sense of Touch and of Temperature.—Tactile stimulation is perceived by the newborn child, or at least is able to produce reflexes. Preyer and, before him, Kussmaul and Genzer, have tested the tactile sensibility in different parts of the body. If a

small rod, a finger or a teat, or something of the sort be put in the mouth of a newborn child it begins to suck it; the entry of nourishment is based on these sucking and swallowing reflexes caused by tactile stimulation. The sucking reflex may often be produced by touching the lips. Stimulation of the nasal mucous membrane by tickling, by acid or ammoniacal vapour, &c., always causes strong reflex actions in the full-time newborn, such as sneezing, wrinkling the forehead, and defensive movements. Secretion of tears also takes place from the nasal mucous membrane in the first days. Attention has already been called to the easily elicited facial reflexes of the young infant. According to Preyer, reflex movements of the nose takes place particularly easily, and of the palmar surface of the hand (grasping of the touching finger) and the soles of the feet; those of the shoulder, chest, abdomen, back and thigh are less easily produced.

The infant usually responds to more drastic stimulation of the skin, such as a smack, with loud screams, thus showing that it is sensitive to pain. During the first few days premature infants do not respond at all to moderate pricking with a needle; full-time ones already react distinctly after the first or second day. Preyer thinks that the compression of the body during birth may produce a feeling of discomfort in the child. That painful sensations are also produced by the internal organs is shown by the obvious expression of feelings of discomfort during the pains of colic. Nevertheless, it is generally accepted that while the reflex excitability for local tactile stimulation in the newborn is even greater than at a later period, the perception of pain is considerably less; that the time of reaction is longer and that the pain, such as in operations, has on the whole not nearly so great an intensity and duration as in the older child.

The sensitiveness to temperature is already present to a large degree in the newborn child. Preyer points out that the stimulation of the heat receptors by the first warm bath after the cooling that follows birth is found to be pleasant by the child, this being recognised by a distinct alteration of the countenance. Even after a few days the expression of satisfaction in the warm bath is quite striking. The stimulation of the respiration by hot baths and cold sponging depends on the marked reflex excitability by thermal influences. The sensibility of the buccal mucous membrane, of the tongue and lips for heat and cold is also strikingly great in many children even in the earliest days.

Cramer describes the newborn child as a "subcortical being," in which all manifestations of life take place unconsciously and in a purely reflex manner; in his opinion the only reflex actions which take place immediately after birth are those which pass through the spinal cord and the medulla oblongata. It cannot be denied that the majority of the above named reflexes started by the stimulation of organs of sense take place without the participation of the cerebrum; the sensation of hunger and thirst and the searching movements performed by the mouth as a result of these

sensations may still be regarded as purely reflex. It can, however, hardly be stated with certainty that no conscious sensibility is present in the undoubted signs of discomfort shown by the child when, for instance, its napkins are wet, or when it has colic, or when its nates are cleaned with a cold damp cottonwool swab, and also in the signs of pleasure which it shows after drinking or in the warm bath as early as the first days of life. It must further be pointed out that infants with cerebral lesions behave quite differently from normal ones; a number of reflexes also take place in these, they suck for instance quite well at the bottle if not at the breast, otherwise they lie quite quiet and still and the cries sometimes of pain and sometimes of anger emitted by the healthy child are conspicuously absent. The idiotic and imbecile child also does not react with the usual cry, a condition which assists the diagnosis even in the first days (Thiemich). The natural disposition of later life or at least of later infancy is often already expressed in the newborn period. Even though the importance of education be fully appreciated the fact must still be recognized that there are children, by nature, both good and bad, some who are always contented from the first day of life onwards and some who are always restless. These marked differences in the psychical condition point to the fact that the newborn child as a rule is not, as is often represented, a reflex being like the anencephalus.

PART II

Feeding

(A) NURSING AT THE MOTHER'S BREAST
UNDER PHYSIOLOGICAL CONDITIONS

Human Milk during the First Part of Lactation

If an attempt be made, shortly after delivery, to squeeze milk out of the breast, a fluid usually spurts out of several pores, which is partly a deep yellow and partly a turbid, sticky, sometimes stringy secretion. This secretion, which is known as colostrum,¹ differs in some respects from the later mature human milk.

These differences, which always vanish after the increase of the secretion and completeness of the evacuation of the breast, partly disappear during the first week, and partly later on. These differences concern, on the one hand, the morphological conditions, and on the other the chemical and physical peculiarities.

The yellow colour which is the most striking peculiarity of the colostrum milk is characteristic of the mammary secretion of the first few days. As soon as the secretion has started the yellow colour diminishes considerably and usually changes at the end of the first week to the white of the mature human milk. The yellow coloration never occurs later in the lactation period, nor does it return at the end of that period. It derives its presence from a yellow pigment which adheres to the fat (Czerny). If the colostrum milk be centrifugalized or left to settle, in the layer of cream will be noticed a secondary division into two layers, an upper zone of yellow colour (colostrum fat) and a subjacent zone of freshly produced white milk fat (Cohn).

The microscopical examination of early milk shows a very different appearance from that of mature human milk. Besides the different fat globules and irregularly formed accumulations of fat drops, the most noticeable constituents present are the so-called colostrum corpuscles. These are large, circular or irregularly formed mulberry-like cells filled with numerous minute drops of

¹ Many consider colostrum to be merely the secretion present in the mammary gland *before* confinement, the quality differing in many respects from the colostrum milk of the *pregnatorial* period.

fat. Opinions are not yet unanimous concerning their origin. The majority of authorities agree with Czerny, who considers them to be leucocytes whose function is to remove the fat from the glands when it is not excreted in the usual way by means of the ducts. In consideration of the infrequent occurrence of colostrum corpuscles in the mature milk the opinion has been given that the corpuscles are not chiefly or exclusively present to absorb but also to bring material for the secreting glandular cells in the parenchyma (Pfaundler). The plentiful appearance of colostrum corpuscles is probably due to a disproportion between the production of the secretion and its evacuation, that is to an obstruction of secretion, such as occurs under physiological conditions during the first days when the gland fills and only a small amount of secretion is sucked out. Others consider the colostrum bodies to be epithelial cells (Popper), which become detached from the epithelial lining of the alveoli or ducts and reach the lumen, where they undergo fatty degeneration. According to Arnold there are both leucocytic and epithelial colostrum corpuscles, the latter being in preponderance. If smear preparations of colostrum milk be treated with alcoholic ether, and if the preparation thus fixed and freed from fat be treated with suitable stains, it will be seen that the larger colostrum corpuscles appear as large mononuclear cells with clear nuclei and the smaller as polymorphonuclear cells with neutrophil granules. Besides the colostrum corpuscles the colostrum milk contains a fairly large number of polymorphonuclear leucocytes, lymphocytes and a large number of the so-called "crescents," cap-shaped formations, staining with nuclear stains, often markedly nucleated, which adhere to larger fat drops. According to Cohn at the beginning of lactation the fresh current of secretion results in a discharge of those cellular elements which had collected in the gland during pregnancy. It is possible, however, that a certain proportion of the cells of the early milk are of quite recent growth and are derived from a migration of white blood cells into the gland spaces during the first days of the puerperium. The different degrees of diapedesis might depend on the variable amount of the secretion obstructed, the large or small supply of blood to the gland, the early or late application of the child to the breast, its power to suck, and on the quick or slow emptying of the breast. The presence of colostrum corpuscles in the early milk is of very short duration. At the end of the first week, if the breast has been properly emptied, the milk is almost always free from cellular elements and has also assumed the microscopical appearance of mature milk.

The numerical proportions of the different cellular elements of the colostrum are shown by Zuckerkandl's following table. The figures are derived from preparations which were made in the following way: 1 c.cm. of milk is centrifugalised, the sediment is sucked up with a fine pipette, and after remaining twenty-four hours in alcoholic ether is stained with hematoxylin-eosin.

PROPORTIONS PER CENT, OF THE CELLS OF COLOSTRUM MILK ON THE DAY OF COMMENCING LACTATION.

| | | POLYNUCLEAR | MONONUCLEAR | | | | | |
|----|---|-------------|-------------|-------------------------|---------|-------------|---|----|
| | | | Ordinary | Colostrum corpuscles | Cleasts | Lymphocytes | | |
| 1 | — | 38 | 14 | 2 | — | — | — | 16 |
| 2 | — | 21 | 21 | — | — | — | — | 8 |
| 3 | — | 66 | 10 | 5 | — | 7 | — | 3 |
| 4 | — | 50 | 15 | 8 | — | 5 | — | 5 |
| 5 | — | 65 | 12 | 8 | — | 9 | — | 6 |
| 6 | — | 64 | 26 | 4 | — | 11 | — | 5 |
| 7 | — | 50 | 14 | 6 | — | 11 | — | 9 |
| 8 | — | 54 | 14 | 10 | — | 7 | — | 8 |
| 9 | — | 54 | 10 | 6 | — | 13 | — | 10 |
| 10 | — | 60 | 22 | 6 | — | 9 | — | 3 |
| 11 | — | 60 | 16 | 5 | — | 13 | — | 6 |
| 12 | — | 55 | 24 | 6 | — | 10 | — | 2 |
| 13 | — | 48 | 12 | 5 | — | 16 | — | 9 |
| 14 | — | 50 | 12 | 10 | — | 9 | — | 8 |
| 15 | — | 36 | 18 | 5 | — | 23 | — | 8 |
| 16 | — | 54 | 24 | 6 | — | 9 | — | 7 |
| 17 | — | 53 | 18 | 14 | — | 5 | — | 14 |
| 18 | — | 52 | 25 | 3 | — | 13 | — | 4 |
| 19 | — | 53 | 21 | 13 | — | 6 | — | 9 |
| 20 | — | 46 | 24 | 7 | — | 20 | — | 13 |
| 21 | — | 46 | 28 | 4 | — | 14 | — | 8 |
| 22 | — | 46 | 20 | 10 | — | 9 | — | 6 |
| 23 | — | 44 | 26 | 5 | — | 16 | — | 9 |
| 24 | — | 32 | 26 | 10 | — | 4 | — | 18 |

Nos. 1—5 very good nurses.

No. 6 a good nurse.

Nos. 13, 15 good nurses.

Nos. 20—23 bad nurses.

No. 24 very bad nurse.

French authorities (Weill and Thévenoz, Lévy) have put forward the opinion that the number of polynuclear cells in the colostrum milk permits of certain conclusions with regard to the productivity of the breast concerned, in so far as a large number of polynuclear cells (over 20 per cent.) should result in a favourable lactation. According to Zuckerkandl's investigation this opinion certainly holds good for extreme cases, but does not permit of any generalization.

The reaction of human milk is amphoteric, acid with phenolphthalein, and alkaline with litmus. The first milk is about half as strongly acid with phenolphthalein, and about twice as strongly alkaline with litmus. From the third day of lactation the reaction remains fairly constant in the same individual (Engel).

The specific gravity of colostrum amounts to 1.052 to 1.060, and is therefore higher than mature human milk (1.026 to 1.036).

Schneef found the depression of the freezing point = 0.549 to 0.595. The highest values are observed on the third and fourth days after delivery.

Allaria found the viscosity of human colostrum to be 4.433 to 1.963 in the first five days after birth, in the next five days 3.802 to 1.838; its viscosity is considerably higher than that of milk (Köppe). We are indebted to Basch for some particularly exhaustive researches on the changes in viscosity of human milk during the first days. He also found a fall in the viscosity and a liquefaction of the mammary secretion during the first six to eight days after birth.

II.

| Time of lactation in days p. p. | Total nitrogen | Fat | Lactose (anhydrous) | Asa | Citric acid | Dry residue | Protein |
|---------------------------------|----------------|------|---------------------|------|-------------|-------------|---------|
| 1 | 0.83 | 2.80 | 3.40 | 0.34 | 0.01 | 11.60 | 2.00 |
| 2—11 | 0.77 | 2.14 | 6.26 | 0.37 | 0.03 | 11.39 | 1.61 |
| 20—40 | 0.20 | 3.87 | 6.43 | 0.22 | 0.05 | 11.35 | 1.10 |
| 50—120 | 0.17 | 2.40 | 6.33 | 0.20 | 0.05 | 11.44 | 1.00 |
| 170 and later ... | 0.14 | 2.65 | 6.88 | 0.19 | 0.05 | 11.85 | 0.81 |

Nitrogenous Constituents.—The N-content of the colostrum is considerably higher than that of the mature milk; it may be more than five times as great.

Birk, in an experiment, found about 0.11 to 0.18 per cent. N in the mature human milk.

| | |
|-----------------------|-------------------------------|
| On the 2nd day | 0.63; 0.41; 0.33 per cent. N. |
| " 3rd " | 0.23 per cent. N. |
| " 4th " | 0.26 per cent. N. |

In another investigation in which the child sucked very badly, so that the colostrous character of the milk was maintained for a longer time, the figures were much higher; the nitrogen content of the successive feeds amounted in percentages to:—

| | |
|-----------------------|--------------------------------------|
| On the 2nd day | 1.9 1.17 1.5 1.5 1.1 |
| 3rd " | 1.0 0.6 0.47 0.05 0.55 |
| 4th " | 0.50 0.48 0.43 0.35 0.37 |
| 5th " | 0.37 0.31 0.34 0.31 0.42 |
| 6th " | 0.34 0.13 0.43 0.16 0.21 |
| 7th " | 0.17 0.25 0.31 0.30 0.48 |
| 8th " | 0.41 |

The following figures from Camerer and Söldner give a few facts with regard to the division of the nitrogen:—

III.

| Time of lactation | Total N | Filtration N | Ur-N | Filtration N-Ur-N | Protein Total N-Ur-N $\times 6.25$ |
|------------------------------|---------|--------------|--------|-------------------|------------------------------------|
| Col. I (26th-28th hour p.p.) | 150 mg. | 35 mg. | 14 mg. | 11 mg. | — |
| — II (48th-68th) | 256 " | 51 " | 11 " | 24 " | — |
| 5th and 6th day | 342 " | 42 " | 10 " | 32 " | 3.37 per cent. |
| 8th-11th day | 256 " | 41 " | 9 " | 32 " | 2.74 " |
| 20th-40th day | 188 " | 40 " | 12 " | 28 " | 1.50 " |
| 70th-120th day | 175 " | 27 " | 10 " | 17 " | 1.53 " |
| 170th day and later | 137 " | 15 " | 12 " | 14 " | 1.25 " |

Total N = Nitrogen as determined by Kjeldahl's method.

Filtration N = Nitrogen filtrate after precipitation with tannic acid.

Ur-N = Urea-nitrogen as determined by Hüfner's method.

As is shown by Tables I and III, the N-content varies greatly in different individuals. With regard to the filtration or residual nitrogen which remains after the precipitation of the protein, this possibly contains somewhat more urea nitrogen, but as a rule it varies little with the increasing lactation. The larger N-content, therefore, depends on a higher protein content. No accurate experiments are available concerning the nature of these protein bodies, and especially with regard to the proportion of casein to coagulable protein. The coagulation of colostrum on boiling indicates that the latter is present in large quantity. A solid coagulation also takes place on the addition of acid. A part of the protein N of colostrum is derived from the numerous cellular elements, but these nitrogenous constituents must not be valued too highly. By mixing 1 c.cm. of watery pus with 100 c.cm. milk Schloss obtained a mixture which, when examined microscopically, showed in each field approximately the same number of leucocytes as of colostrum corpuscles in colostrum; in this experiment the N-content of the pus was only about 4 per cent., so that the increase of protein in the milk was but small.

At first the protein content falls fairly rapidly, but at a later period of lactation it again shows a tendency to fall.

The sugar content of colostrum is low as compared with that of mature milk, this being generally attributed to reabsorption owing to obstruction of the secretion. The absorbed lactose is the cause of the lactosuria of the woman during confinement. In contrast with the protein content the sugar content rises with the progress of lactation.

With regard to the fat content of colostrum milk it is not possible to make a definite statement, since during the first days only the first portions of the still scanty secretion come to be analysed; sufficient quantities of mixed milk are not at our disposal. In the estimation of fat in milk it is necessary to consider the time of the taking of the milk, and this is seldom possible.

Höbelfeld found a gradual fall in the amount of fat in the milk of goats during lactation, this fall being particularly steep during

the first days. Such a fall does not appear to take place in the human being. (See Tables I and II, pp. 82-3.)

With regard to the quality of the fat of colostrum, Eichelberg and Engel were able to show a higher oleic acid content to be the rule; this is shown by the considerable higher iodine value. In respect of this amount, in most women there is a tendency to decrease at the beginning of the second week, and yet up to the end of this period not a single normal average of 40 to 50 was observed in thirteen cases. It is interesting that the iodine value of the fat of the colostrum (62) is identical with that of the human body fat. Engel considers that at the beginning of lactation only body fat, and with increasing amount and duration of secretion, nutrition fat also, is carried to the mammary gland and passed out.

| Nurse I. | | | | Nurse II. | | | |
|--------------------|--------------|----------------|--------------------------|--------------|----------------|--------------------------|--|
| Day after delivery | Iodine value | Amount of milk | Remarks | Iodine value | Amount of milk | Remarks | |
| 3 | 61 | — | In the maternity clinic. | — | — | In the maternity clinic. | |
| 4 | 61 | — | | — | — | | |
| 5 | 62 | — | | 62 | — | | |
| 6 | 64 | — | | 61 | — | | |
| 7 | 65 | — | | 65 | — | | |
| 8 | 57 | — | | 63 | — | | |
| 9 | 59 | — | | 50 | — | | |
| 10 | 60 | — | | 64 | — | | |
| 11 | 56 | 965 | | 61 | — | | |
| 12 | 44 | 945 | | 60 | — | | |
| 13 | 41 | 950 | As nurse in a situation. | 61 | 478 | Nurse. | |
| 14 | — | — | | 48 | 888 | | |
| 15 | 48 | — | | 40 | 1,000 | | |
| 16 | — | — | | 48 | 930 | | |
| 17 | 41 | — | | 46 | 1,310 | | |
| 18 | — | — | | 45 | 1,000 | | |
| 19 | 46 | — | | — | 1,180 | | |
| 20 | — | — | | 51 | 1,200 | | |
| 21 | — | — | | 45 | 1,150 | | |
| 22 | 43 | — | | — | — | | |

The fall of the iodine value appears to depend on the rising efficiency of the breast. When the establishment of lactation takes place suddenly the iodine value falls almost as rapidly to the normal value. Under ordinary conditions this peculiarity of the colostrum appears to outlast the others considerably.

The early milk is relatively rich in lipoids; these accumulate in the blood during pregnancy, and are excreted during the puerperium chiefly in the mammary secretion (Herrmann and Neumann).

A parallel exists between the total ash and nitrogen in so far as both amounts diminish gradually during the course of lactation. According to Schloss, the colostrum differs from the mature milk in that the alkaline earths are in small proportion compared with the alkalis. The lime and phosphorus values in colostrum milk are much smaller in proportion to the total nitrogen than in mature milk. Correspondingly the proportion of phosphoric acid is small as compared with that of choline. (According to the analyses of

Birk, the P_2O_5 content of colostrum is higher.) Moreover, the relatively high sodium value of the colostrum milk is noteworthy. The proportion of the three most important cations, Ca, Na and K, is absolutely different from that of mature milk. The proportion of Ca in the colostrum milk barely amounts to a seventh, while later it is a third of the total. The lime values undergo a sharp rise at the beginning of the lactation period, until in the mature human milk a fairly constant value is maintained. Mineral analyses show that colostrum resembles blood serum much more closely than does mature milk.

| | Colostrum (Schloss) | Colostrum (Birk) | Transitional milk (Schloss) | Mature milk (Schloss) |
|-------------------------------|------------------------|---------------------|--------------------------------|--------------------------|
| N | 2.350 | — | — | — |
| Total ash | 5.048 | 2.864 | 2.331 | 1.839 |
| CaO | 0.3350 | 0.250 | 0.400 | 0.3785 |
| MgO | 0.0680 | 0.093 | 0.0680 | 0.0857 |
| Na ₂ O | 0.3424 | 0.544 | 0.3578 | 0.1880 |
| K ₂ O | 0.2048 | 0.220 | 0.0780 | 0.5021 |
| P ₂ O ₅ | 0.1804 | 0.137 | 0.5933 | 0.4026 |
| | (0.0933) | — | — | — |
| Cl | 0.8047 | — | 0.4454 | 0.3055 |
| | (0.7622) | — | — | — |

100 gm. of total ash contain (Schloss):—

| | Colostrum | Transitional milk | Mature milk |
|-------------------------------|-----------|-------------------|-------------|
| CaO | 11.06 | 18.79 | 19.62 |
| MgO | 2.46 | 3.12 | 4.71 |
| Na ₂ O | 10.00 | 15.31 | 10.16 |
| K ₂ O | 28.37 | 30.72 | 26.60 |
| P ₂ O ₅ | 12.48 | 17.51 | 22.23 |
| Cl | 31.82 | 19.28 | 15.79 |

With regard to ferments it was proved that peroxidase and reductase are only present in colostrum (Hecht), and catalase is usually much more plentiful in early than in later milk. The catalase content is usually inconstant, nevertheless a fall in the catalase value to a lower amount takes place during the first three to five days (van der Velden); at all events, the cell content of the colostrum milk may be a factor involved in this connection.

With regard to the immune bodies which pass over from the blood serum to the milk there are as yet no comparative researches for human milk concerning the differences between early and mature milk, but it should be permissible to apply to human beings the results of animal experiments concerning the passage of anti-toxins through the milk. Ehrlich and his pupils were able to show that the milk antitoxin is combined with the genuine hemialbumose (lactalbumin and lactoglobulin). Since the amount of coagulable protein passing over at the beginning of lactation is considerably greater than at a later period, it may be concluded that the colos-

trum milk also contains more antitoxin. According to Engel, "it is almost universally agreed that all biological peculiarities of milk are much more in evidence in the colostrum than in the mature milk. We see this in the immune bodies, the amount of which falls progressively during the first days of lactation, and also in the enzymes that have been carefully studied. A still more exhaustive study of the enzymes and other biological qualities of milk will probably give an insight into the physiology of the secretion of milk protein, since the substances mentioned chiefly originate from the blood serum, thus giving us a certain indication. It is already possible to see a closer relationship between colostrum and blood serum than between blood serum and mature milk."

The Meaning of Colostrum Milk

The points of difference between colostrum milk and mature human milk may be summarized as follows: Colostrum milk is more concentrated and more viscous, it contains more protein bodies, and among these a relatively larger proportion of coagulable protein; it is poorer in milk sugar; the fat exhibits fewer perceptible differences in quality, but differs from the fat of mature milk by containing more oleic acid and also by the yellow pigment allied to fat; colostrum milk is relatively rich in lipoids, its ash content is much greater, and with regard to the individual salts there are noteworthy differences as regards the relative scarcity of alkaline earths as compared with the alkalis; the amount of enzymes as well as that of the immune bodies possibly present is higher.

A large number of the differences between colostrum milk and the mature product may be simply explained by the fact of re-absorption of milk at the same time as production in the colostrum period (Czermy-Keller), e.g., the relative poverty of water, the low milk sugar content, accompanied by lactosuria, the occurrence of colostrum corpuscles, which perhaps have the function of taking the obstructed milk fat out of the mammary gland. Nevertheless, there is much evidence to show that it is not merely an alteration in concentration occurring in the course of lactation, but that a complicated correlated displacement is present (Sedlitz). It is but necessary to consider the enormously high protein content, the preponderance of coagulable protein, the differences in the composition of the fat, and the results of inorganic analyses. It must also be borne in mind that the alterations in the milk do not by any means take place with regularity, that certain properties of colostrum sometimes disappear very quickly, others taking much longer, and that the secretion, known as mature milk, is really established quite gradually. It is therefore quite certain that the early milk cannot be regarded simply as concentrated milk.

Has the colostrum a specific importance for the newborn child? Is it particularly advantageous for the newborn child to be fed on the mammary secretion corresponding to the date of its birth—that

is, first with colostrum, then with transitional milk, and finally with the mature human milk? It is difficult to reply to this question, since from the clinical point of view we have really no evidence to show that worse results follow the feeding of a young infant on human milk belonging to a later period of lactation, correct technique being assumed. From the theoretical point of view preference must be given to the natural processes of nutrition. Hohlfeld compared the results of nutrition in newborn animals which had been fed on colostrum and on mature milk of their own species, and found with goats that those fed on colostrum were distinctly ahead in the first week of the experiment, whereas with dogs and guinea-pigs no essential difference could be seen. In the high protein and fat content of the colostrum he sees the fulfilment of a physiological demand, since the heat loss of the newborn requires increased heat formation. This can only be obtained from mature milk by drinking larger amounts. The advantage of colostrum, therefore, is that it can satisfy the larger demands of the organism with smaller amounts than mature milk. The results of experiments on metabolism also support this view (see pp. 14, 19). It is certainly not unimportant that the alterations in the secretion of the mammary gland pursue a course parallel with its output; that, e.g., in premature infants with weak powers of sucking the concentration, and with it the caloric value of the nourishment, reaches a higher level on account of the small amount of milk taken. There is thus an equilibrium between power of sucking and amount of fluid drunk on the one hand and alterations in the secretion on the other. It is also difficult to prove the importance of the transference of immune bodies from the mother to the child, in cases in which these are plentiful in the mammary secretion during the first days. Birk refers to the researches of Rauterisen which showed a close resemblance in biological reactions between the protein of the colostrum milk and the serum protein of the mother, and that the nourishment with colostrum milk should be considered somewhat as a continuation of the umbilical nutrition in the newborn child. "It is the transitional stage between placental nourishment and nourishment with mature milk. It is also possible that in the newborn child a peculiar condition is present in so far as that it is able to absorb the protein bodies of the colostrum unaltered and unhydrolysed from the alimentary canal."

From the practical point of view the superiority of colostrum over the human milk of a later period of lactation lies above all in the fact that the former, taken in smaller quantities by the child, represents a diet of relatively high caloric value; the other advantages of colostrum, e.g., in the choice of a wet-nurse, are hardly of practical importance.

A mildly laxative action is often attributed to colostrum. It is, however, not very probable that the frequently occurring symptoms of intestinal irritation at the time of the change from

meconium to milk stools should be primarily due to the colostrum character of the milk. The view that colostrum milk is more laxative than the mature product is not much in accordance with our more recent views as to the action on the intestine of artificial foods relatively rich in proteins and poor in sugar; the higher concentration itself must then be the origin of such an action. That human milk, as compared with other forms of milk, is usually more likely to cause peristalsis and intestinal irritation on account of its relatively high sugar content is undeniable, but it has not been proved that early milk has a purgative action as compared with mature milk. Czermy and Moser found that the motions of an infant fed on colostrum assumed a green or brown colour which immediately changed to the normal yellow colour as soon as the child again received the usual human milk.

The Causes of Lactation

During pregnancy the mammary gland undergoes an important increase of its parenchyma. During the last part of pregnancy there is already a secretion of a colostrum-like fluid. True lactation, however, is not established until after confinement. In this connection it is of little importance whether birth follows after a normal duration of pregnancy or whether this is brought to an early termination. H. Cramer describes the case of a girl who, after an abortion in the eleventh week of pregnancy had so plentiful a secretion of milk that she was able to act as wet nurse for five months. On the other hand it does not seem possible to produce a true secretion of milk before confinement and independently of pregnancy. By regular application of a strong child to the breast of a woman in the last week of her second pregnancy, Hildebrandt attempted to cause the mammary secretion to begin. This succeeded in so far as considerable secretion of watery fluid resulted, it was not until the third day after delivery, however, that the secretion assumed the characteristic appearance of milk. In opposition to this view it has been stated by many that in a pregnant woman it is possible to produce lactation simply by regular sucking at the breast and without interfering with the progress of pregnancy (Seilheim). Cramer even presumes that this may occur in a virgin during menstruation. Be that as it may it is none the less true that delivery even before the normal end of pregnancy always results in mammary secretion and this certainly occurs also without its being necessary for the function of the organ to be stimulated by sucking.

Three essential factors are involved in lactation:—

1. The gland rudiment.
2. The factor causing increase in the parenchyma (during pregnancy).
3. The factor liberating the secretion (after confinement).

According to their anatomical structure breasts may be divided into two classes, those rich in connective tissues and those in which

parenchyma preponderates. The parenchyma of the gland may be insufficiently developed in cases in which there is simultaneously full development of the connective tissue part of the mamma (Engel). When the parenchyma is rudimentary, powerful growing impulses may not be responded to because the breast still remains poor in parenchyma after the confinement. It is possible that a gland which is not a priori poor in parenchyma may undergo for a long period a sort of degeneration through inactivity, a stimulation to growth caused by pregnancy will then be insufficient to cause the full functioning of the organ. In this way the frequent difficulties of lactation met with in elderly primiparae may be explained.

Not only a theoretical but also a practical interest is centred in the results of researches concerning the origin of the hyperplasia of pregnancy of the mammary gland and its secretion following confinement. They form the basis for the attempts, hitherto somewhat unsuccessful, to find a means by which an imperfectly functioning gland may be made to secrete and maintain its secretion.

We will not discuss here the various theories of lactation but confine our attention to the most important facts determined by experiment and clinical means. It may be considered as established that the hyperplasia and secretion of the mammary gland are dependent on the processes in the genital tract of the pregnant woman and that both growth and secretion are thereby stimulated. The point now to be considered is the way in which this stimulation is transmitted and the particular organs from which it originates.

Concerning the way, it may be regarded as certain that the activity of the mammary gland is not aroused by nervous stimulation proceeding from the genitals; and it may also be excluded that growth and secretion are merely dependent on the quantitative alterations in the blood supply. The origins of such processes are much more closely connected with qualitative chemical changes in the blood. It may thus be the case of the occurrence of stimulatory and inhibitory substances or of particular materials of construction and nutrition in the blood (Pfaundler).

The stimulating substances (hormones) which should cause lactation have a double function to fulfil; hyperplasia of the parenchyma and stimulation of the secretion. Since no secretion takes place during pregnancy both functions must originate from different hormones, or if there be but one hormone the secretory component must be inhibited during pregnancy. There is, however, no antagonism between the two functions since the hyperplasia also continues till the time of milk production.

The organs of internal secretion concerned are the ovary and the placenta; also the hormones may arise from the foetus itself. With regard to the part attributed to the ovary there is agreement in so far as it is not generally regarded as the place of origin of the body which stimulates secretion. According to Grünbaum the removal of the ovary is followed by the secretion of milk, so that in this respect the generative gland possesses an inhibitory function.

Opinions differ as regards the functions of the ovary as the site of the formation of the substance causing growth. The ovary alone can be involved in the growth impulses at the time of puberty and menstruation. With regard to the hypertrophy of the mammary gland during pregnancy Halban is of opinion that the ovary is not of importance, its protective function being taken over by the placenta, or by the trophoblasts of the chorionic epithelium. Basch, on the other hand, thinks that the growth of the gland in pregnancy is due to stimulating bodies derived from the ovary after fertilization. Fertilization does not appear to be absolutely necessary in this connection, since Aschner and Grigoriu were able to produce hypertrophy of the mammary glands even in virgin animals by injecting foetal and placental extracts. It may be considered as an established fact that the ovary certainly contains substances which have a stimulating action on the growth of the mammary parenchyma, but that these hormones may also be formed in the placenta and in the uterine mucous membrane, so that the function of the ovarian hormone may be supplanted by growth impulses derived from the ovum (placenta and foetus).

Only the placenta and foetus are involved in the discharge of the milk secretion. Since the secretion of the glands does not begin till after the birth, that is after the extrusion of the ovum, Halban concludes that those substances in the placenta which promote growth during pregnancy, have at the same time an inhibitory action on the process of secretion; this disappears with the extrusion of the placenta. The ensuing secretion is maintained and increased by the sucking stimulus of the child, or by the evacuation of the mammary gland generally; it ceases, however, as soon as the gland ceases to be utilized. The factor liberating the secretion of milk which proceeds from the placenta is, according to this theory, not a positive but a negative one. Similar to this is the opinion of Hildebrandt, who considers that the placenta provides an internal secretion which protects the gland from that autolytic disintegration which, in his opinion, takes place in the secreting gland.

The investigations which prove that the injection of placental extract may actively stimulate the secretion of milk contradict the theory which attributes the secretion of milk to a removal of inhibitory substances from the placenta. The hormone which causes the secretion of milk has been called by Basch placenta-secretin (after Starling). According to Lederer and Przibram intravenous injection of fresh placental extract in definite concentration produces in the goat an increased secretion of milk, which begins after three to ten minutes and disappears half to two minutes later, enormous amounts being attained when larger injections are given. Aschner and Grigoriu were able to produce true milk secretion not only by injection of placental extract, but also by that of foetal extract apart from hypertrophy of the mamma even in the virgin animal. These authorities also attempted to obtain

insight into the nature of the substances causing lactation, and were able to show that in an animal which has once given milk, but is dry at the time of the experiment, the subcutaneous administration of any lymphagogue is sufficient to produce definite secretion of milk, but that in the virgin animal non-specific means did not succeed in producing lactation.

Against the hormone theory Pfaundler points out that the nutrition theories originally formulated by Rauber must not be dismissed as incorrect. These are based on the belief that after extrusion of the ovum a nutritive material becomes available which may serve for milk formation after birth. Pfaundler thinks that the withdrawal of certain nutritive substances by the genital glands, embryo and ovum temporarily disturbs the equilibrium of physiologically active substances in the blood, and that the antagonists of these substances (hormones of other authorities) find specific receptors (affinities) in the other organs of the generative system. The hyperplasia of the mamma and analogous phenomena of pregnancy are thus due to these antagonists.

As appears from the above we are still frequently in the realm of hypothesis with regard to the nature of the process of lactation. Experiments have only shown that the growth of the breast is influenced by the ovary, and that impulses causing both growth and secretion are derived from the placenta and the foetus. A fully satisfactory explanation has not yet been given for the fact that the secretion does not develop until after the expulsion of the foetus.

The Mammary Secretion and Suckling

The true mammary secretion never begins immediately after delivery. On the first day the consistence and filling of the breast are hardly different from the condition in the last period of pregnancy. If an attempt be made to express secretion with the hand or to draw it off with a pump drops of secretion will flow out of one opening or from several pores. These are either turbid, cloudy and frequently viscous, or of the typical yellow colour of colostrum. Often a watery secretion passes out of one opening and a colostrous secretion out of another. The amount of secretion which can be expressed is usually small; only comparatively rarely, and especially in multiparae who have already nursed, does the secretion sometimes spurt out in a stream, so that larger amounts (several cubic centimetres) of fluid may be pressed or sucked out.

The secretion may now take place in one of two ways. In a number of cases the parenchyma of the gland fills gradually. The breasts become somewhat larger, the woman usually experiences a slight stretching sensation, and milk may now be expressed without difficulty, it may flow out in plentiful drops or spurt out in a stream. The child, which perhaps has hardly drunk a weighable amount during the first attempts at sucking, now shows a distinct difference in weight before and after feeding. The period of transition to true

secretion usually falls on the second day; this is established under normal conditions on the third or fourth day. The secretion usually loses its colostrous character about this time and assumes the appearance of milk, and is usually fairly concentrated.

In the second type the beginning of secretion is fairly sudden and more precipitate. This may be described as a gush of milk ("Milcheinschuss"). The breasts become hard, turgid, and often markedly increased in volume, the skin over the gland is shiny, stretched, and frequently shows a bluish network of veins shining through. The woman complains of considerable feeling of turgidity and pain, and is sensitive to the slightest pressure. This condition is most frequently found on the third or fourth day p.p., sometimes already on the second day, and normally lasts no longer than one to two days. The duration of the hardness is chiefly dependent on the amounts of fluid evacuated.

There are many intermediate transitions between these types. The establishment of lactation may also take place gradually, the hardening of the breasts may be imperceptible and may only last a few hours. It may also affect only a few parts of the breast, usually the outer parts of the base. To the touch the filled mammary ducts may frequently feel like hard cords. The softening often does not follow in a regular manner, but in such a way that hardness persists longer in certain parts of the breast, or that one breast remains harder than the other.

All of these processes are usually finished by the end of the fourth day. The gland is then in a secreting condition, although the quality and quantity of the milk secreted at this time undergo manifold variations in the course of the ensuing days and weeks.

Variations are present not only as regards the form of the beginning of secretion, but also in the manner in which the gland is emptied. It is usual to speak of breasts which secrete easily or with difficulty, but it is better to use the terminology proposed by Schlossmann, and describe them as yielding (*gebend*) with ease or with difficulty.

There are mammary glands from which large amounts of milk may be obtained as easily by manual pressure or suction pump as by the sucking of a child with little power of suction; glands from which secretion flows out spontaneously, and, on the other hand, those from which only relatively small amounts can be obtained even by a powerfully sucking child. These differences by no means always correspond to the plentiful supply of the milk; since there are certainly glands rich in milk, but at the same time yielding with difficulty, especially in primiparae with tight breasts; in such cases difficulties of lactation are not infrequent with somewhat weakly sucking infants without there being any fault to find with the productivity of the breast.

The cause of these differences in secretory ability is not definitely known. According to Cramer, the ease or difficulty of the yield depends above all upon the milk content at the time. As opposed to

this, Schlossmann considers the amount of production to be to a great extent independent of the power of resisting evacuation. Pfaundler thinks that the determining factor for the form of evacuation might possibly be sought in the co-ordination of all the involved muscular action and in the patency of all the reflex paths.

It is true that a breast yielding with difficulty can hardly be changed during the course of lactation into one which yields easily; none the less, considerable alteration may take place in this direction. From the expressibility of a breast and from its test it is not always easy to decide whether its yield be easy or difficult. A child is often able to suck out large quantities of milk without its being easy to squeeze or pump out milk. This depends, on the one hand, on the superiority of the sucking of the infant over all our methods for the artificial removal of milk, on the other hand, however, it is due to certain nervous influences, inhibitions, which become particularly noticeable during artificial evacuation. It can hardly be denied that psychical influences are also concerned in the function of nursing, especially in the first days while the secretion is being established, if as frequently happens the mother's ability or disability to nurse her child is being decided. Most authorities are of opinion that the ordinary galactagogues and various dietetic measures employed to promote lactation are chiefly of value, if they act at all, by instilling new hope into the woman who doubts her ability to suckle. It is an indisputable fact that goodwill and an encouraging environment are not without influence on the mammary function.

From the differences in the establishment of lactation and from the peculiarities of the maternal breasts alone there result the greatest variations in the process of nutrition, and thereby in the development of the child in the first period of life; variations which we are, however, justified in including for the most part under the heading of normal processes.

We shall deal more fully with the various peculiarities of the nipples, the differences in the qualities of the breasts, the delayed establishment of lactation, &c., when we are describing the difficulties of lactation.

In the newborn child the act of sucking is performed chiefly by the muscles moving the mandible. Whereas in the adult sucking is chiefly performed by the freely movable tongue, in the infant this remains passive on movements of the mandible. This appears to be ordained by Nature, as the greater part of the floor of the mouth is occupied by the wide fleshy root of the tongue, while the short freely movable part of the tongue is made practically immovable by the *frenulum linguae* which extends so far forwards (Escherich). On the border of the gum of the mandible, and somewhere about the region of the future canine tooth, a membranous projection is found on both sides connected with its fellow of the opposite side by a membranous ridge, 1 to 2 mm. high, the so-called

fold of Magitot. It assists hermetic closure of the oral cavity during suckling (Cramer).

According to Basch sucking takes place as follows: At first the mouth of the child encircles the nipple. This reacts with a contraction of the areolar muscle and an erection of the papilla; it comes longer and thinner. This process may be demonstrated at any time by compressing the base of the nipple lightly with the fingers. On account of the rarefaction of the air caused by the downward movement of the mandible the nipple is sucked deeper into the mouth. The jaws are then approximated so that pressure is exerted on the sinus lactiferi. The pressure exerted by the jaw by which milk is expressed from the sinus was investigated by Basch with a dynamometer and found to amount to 200 to 300 gm. in the normal newborn, and to rise in the course of the first two weeks of life up to 700 to 800 gm. The previously stiff nipples are made somewhat softer by the pressure exerted on them by the jaw. Should this cease erection again occurs. The aspiration of the child acts in such a way that more milk runs into the reservoirs of the nipple just after they have been compressed.

Basch thinks that breasts yielding easily and with difficulty differ from one another through the degree of pressure required to overcome the tonus of the muscle of the nipple. He attributes an essential importance to the pressure exerted by the jaw in the region of the areola, and does not consider that the power of aspiration is alone sufficient to suck out milk. The negative pressure which is necessary to aspirate milk from the breast usually considerably exceeds the suction pressure which can be measured by the sucking movements of the infant. Cramer, however, was able to show with a manometer that the child, by means of a series of sucking movements, was able to increase progressively the negative pressure, so that the maximum suction pressure that the child is able to exert amounts to 30 to 140 cm. water; the negative pressure in one sucking movement only amounts to 4 to 14 cm. In the cases investigated by Cramer the power of aspiration exhibited by the child corresponded to the negative pressure which was necessary to suck milk from the breast. It is none the less necessary to assign a definite importance as an accessory factor to the pressure exerted by the jaws, and especially to regular sequence of increase and decrease of the same, since in this way the filling of the evacuated lactiferous ducts is facilitated.

Technique of Suckling

With regard to the preparation of the breast for suckling, it is particularly the nipples which so often demand preparatory treatment. We shall deal later with the abnormalities of the breast before and during pregnancy and possible precautions, also as to how the former may be prevented or corrected. At this point it only remains to be pointed out that the pulling out of flat or depressed nipples during the last months of pregnancy is not to be

recommended in view of the connection between sucking movements at the mammary glands and contractions of the uterus. Such procedure is particularly inadvisable, all the more so as it is of little use in improving the breasts. To prevent the occurrence of fissures, which not infrequently occur when the child is applied to the breast, it is often recommended to harden the nipple during pregnancy by washing, rubbing, or even brushing the nipple and areola with alcoholic lotions. Such treatment may be harmless in moderation, but whether it is of much value is another question. Even after "hardening," most scrupulously carried out, rhagades frequently occur during confinement.

It is beyond our power to prepare the parenchyma of the gland itself for lactation. Anatomical peculiarities such as a relative richness of connective tissue cannot be corrected. The increase in the parenchyma and the formation of the secretion result from internal causes which we do not yet sufficiently understand to be able to make use of them in practice. A good and suitable diet for the pregnant woman is to be recommended in the interests of lactation. But for a generally healthy person it can hardly be of essential importance for the development of the mammary glands.

Also after delivery, on the establishment and after the beginning of lactation, the nourishment should be sufficient but not too much. The intake of fluids should correspond to the woman's sensations of thirst. The inordinate drinking of milk, or even beer, frequently recommended to mothers in the interests of lactation, is certainly not to be advised. If a woman is accustomed to take very little or no alcoholic drink she should under no circumstances forsake this most praiseworthy habit during lactation. On the other hand, it can hardly be advisable to make special attempts just at the time of commencing lactation. It is by no means proved that the alcohol drunk by the mother (naturally within definite limits) is injurious to the child, and the suggestive effect produced by a glass of beer may possibly do more good than the alcohol in it does harm. In general it is quite sufficient for the fluid requirements (which are fairly large) of a nursing woman to be satisfied by water.

Underfeeding is to be avoided during confinement just as much as overfeeding during confinement. Czerny and Keller describe it as a bad habit to limit on purpose the food in the puerperium. They think that in many cases a deficient secretion of milk in the first days p.p. is to be attributed solely to underfeeding.

No particular instructions are required with regard to the quality of the food. The usual hygienic and dietetic precepts also hold good in this connection. Most paediatrists have come to the conclusion that when these are observed the diet of the mother has no influence on the alimentary canal of the infant or on its thriving. The observations that sour dishes and those provoking flatulency, fruit, &c., are injurious to the child will not bear careful criticism.

It must be admitted, however, that certain substances which can be detected chemically do pass over in the milk, and that the

transmission of substances difficult or impossible to detect chemically lies within the bounds of possibility.

The excretion of drugs in human milk has been often studied, and it has been shown that positive results follow therapeutic doses of the following substances (Bacura):—

Iodine (Fehling, Stumpf, van Italle, Bacura).

Salicylates (Fehling, Páuli).

Ether (Gottsch-Besner).

Mercury (Hambarger by introduction of suppositories, Bacura by internal administration of calomel).

Antipyrin (Pinnaut, Ficuz).

Aspirin, arsenic, sodium bromide (Bacura).

While Thiemich regards the excretion of drugs in the milk as small in amount, Bacura is of opinion that some care is necessary in administering drugs to the mother for the sake of the child. According to H. Meyer and Gottlieb (*Experimentale Pharmakologie*) morphine passes over in the milk, and owing to the extreme susceptibility of the infant for this drug the intake of morphine by the mother makes the poisoning of the child a possibility. The fetus or *altero*, which cannot breathe independently, is very resistant towards morphine; the administration of morphine during pregnancy is therefore safe, only shortly before delivery does it endanger the respiration of the child.

Finally, with regard to the transference of true toxins in the milk, for many reasons these can hardly be considered as of practical importance. The bacterial toxins which may circulate in the blood of a sick mother and which may pass over in the milk are usually present in such small amounts that they are of no importance for the child; should the illness of the mother be so severe that a greater toxæmia is present, then the milk will certainly not be used at all. The possibility of transference of bacterial toxins in the milk is still more reduced by the fact that these have had to reach the body through the alimentary canal. They are here subjected to the action of the gastric juice which inhibits or destroys toxins and which is already present in the newborn (A. Schütz). Moreover, as high molecular bodies allied to the proteins they cannot immediately be absorbed; even if the intestinal wall of the newborn animal be permeable to bacterial toxins (tetanus toxin, Effenheimer) the amounts which can pass over from the human milk, under any circumstances, are so small as to be of no practical importance. Vegetable poisons, which in the cow may be excreted in the milk (abrin, ricin, &c.) have nothing to do with natural nutrition. The poisonous substances not of bacterial origin such as may pass into the milk in erysipelas, sepsis, &c., are possibly of greater importance; this question will be more closely dealt with under the contraindications of nursing.

The first application of the child to the breast must take place in a position essentially less comfortable than that usually taken after confinement. It is well known that suckling is most easy when the mother sits on a low stool or on the backwardly tilted seat of a "nursing chair" (*Ammensessel*), which permits of the child being placed on an easily raised knee and for the breast to be brought to the child from above by a slight forward movement of the thorax. Difficulties which are not to be underestimated are liable to occur when the first attempts at suckling take place in a horizontal position, especially when the mother is not able to move much after delivery, and when a primipara is concerned who does not know the technique of suckling.

When the child is to be applied the mother turns the upper part of her body somewhat towards the side which is to be used. The child lies parallel to her body without the head being any nearer than the legs. The head also should lie as far as possible in the

axis of the child's body and not bent forwards as this disturbs swallowing. Care must be taken to see that the mother lies in an extended position and not with her knees bent so that the child has room. For this purpose it is also advisable to put the child to the breast without a pillow, since voluminous wrappings make a successful application much more difficult. The head of the child should be placed so that its mouth comes opposite the nipple, or that the hanging breast of the mother can easily be brought into position; a flat cushion or a folded cloth may be placed under the head of the child. Care must also be taken to prevent the nose of the infant being covered by the parts of the breast near the nipple; this is very liable to occur with flat nipples and wide areolæ and with voluminous breasts, and interferes with the child's sucking. While in the sitting position the breast is directed by the hand of the same side. It is necessary in the lying position for the hand of the opposite side to be used. The part of the breast adjacent to the areola is taken between the index and middle fingers, and the nipple, together with the neighbouring part of the areola, pushed into the child's mouth. The index finger presses lightly on the breast in order to keep the nostrils free.

As soon as the condition of the mother permits, nursing may be undertaken with raised thorax either in such a way that the mother supports herself on the elbows or takes a sitting posture in bed. Care must always be taken that suckling takes place in a position which can be maintained by the mother for a long time without fatigue.

Before the first suckling the breast must be cleaned thoroughly, but with all necessary gentleness. This may be done either with water and soap only or also with 70 per cent. alcohol, and finally with boric solution or boiled water. Such cleansings, carried out with proper care, are also to be recommended during the following days. It is also very important that the hands should be frequently and thoroughly cleaned (including the nails), since these are continually in contact with the breasts. Particular attention must be given to this point in lying-in hospitals, where there often remains much to be desired with regard to the cleanliness of the women who come to the institution immediately before confinement.

Opinions differ with regard to the necessity for the cleansing of the nipples before each meal. A thorough disinfection is scarcely possible, since a powerful antiseptic would be liable to irritate the sensitive tissues of the nipple, and there is always a certain danger that the remains of the disinfectant may reach the child's mouth, e.g., corrosive sublimate (Czerny-Keller). Frequent rubbing may also give rise to mechanical lesions. It is very customary to dab the nipples with a swab soaked in boric lotion before each meal, a harmless procedure, though of doubtful value. According to the experience of Jaschke it may be omitted without disadvantage. If the skin of the nipple be intact it is not necessary to cover it

with ointment. In general, the usual cleanliness observed by clean people at other times than during pregnancy and confinement suffices—cleanliness, of course, that will satisfy all requirements. Clean body linen is also necessary; it is also advisable to put a clean cloth or a piece of absorbent gauze over the breasts during the intervals between meals, this being especially necessary in cases of galactorrhoea, since the wetting of the clothing and bed may be highly unpleasant for the mother.

When, at the time of the establishment of lactation, the breasts become heavy or feel stretched, it is sometimes advisable to fix them with a fairly tight bodice. Should the discomfort only be great at the time of commencing lactation the breast may be fixed and lightly compressed by a bandage (*suspensorium mammae*). Cooling compresses containing a weak solution of aluminium acetate are often helpful. (The skin of the nipple may be protected from laceration by a little ointment.) The best procedure in such cases is always to evacuate and discharge the breasts as far as possible; this can either be done by putting the child to the breast, or by a milk pump if it sucks badly on account of the hardness of the breast. At all events, it cannot be expected that much milk will be obtained from a hard breast with a pump; the yield is often very small, but even a small discharge of the organ is desirable. Manual evacuation is usually impossible in such cases on account of the sensitiveness to pressure.

The buccal cavity of the infant cannot be made aseptic any more than the nipple can be completely disinfected. Such cleansings have been discouraged since the sucking movements alone completely remove the remains of milk, and should the mouth be swabbed out injurious results may follow from mechanical injury to the mucous membrane which facilitates the formation of a bacterial focus (see p. 226). Only when vaginal secretion has entered the child's mouth during delivery is a careful swabbing out immediately after birth permissible and even advisable.

Excessive bacteriophobia gave rise to the suggestion that infants should never be directly applied to the breast during the first few days, but should be made to suck through a nipple shield, in order to prevent infection of the child and of the mother, and in particular the occurrence of mastitis.

This procedure had to be abandoned as the progress of nutrition under these circumstances was not satisfactory (Himmelheber). It must be stated emphatically that suckling with a nipple shield can never take the place of the sucking mechanism at the breast, and that even the best nipple shields yield an amount of milk which is always considerably less than that obtained by direct application. The nipple shield is a most useful instrument under certain circumstances (see pp. 118-120), but under normal conditions its use as a prophylactic against infection can hardly be justified.

Most infants sleep during the first hours of life and show no signs of hunger. Should they be awakened they usually fall asleep again at once. In the majority of cases this condition lasts the

whole of the first day. The rule that a child should not be fed during the first twenty-four hours may therefore be laid down with confidence (Czerny—Keller). Should the child be restless during the latter half of the first day a little water or weak tea may be given, and for the sake of precaution sweetened with saccharin, and not with sugar.

The amount of fluid drunk at this time is usually very small; the infants are usually perfectly satisfied with a little (10 to 20 c.cm.). It is immaterial whether the fluid be given with a spoon or from a baby's bottle.

The rule that the child should not be put to the breast during the first twenty-four hours need not be regarded as an irrevocable law. As a matter of fact it is quite immaterial whether the child be fed during the first day or not (Jaschke); the amount of milk present in the breast and drunk by the feebly sucking infant is usually so small that it does not affect the scales. It may therefore be said that the putting of the child to the breast before the second day is neither necessary nor injurious. For the mother, at any rate, it is pleasanter to have a whole day in which to recuperate after the toils of delivery; as regards the establishment of lactation, it matters little whether attempts at suckling be made as early as the first day, since these are in any case fairly unproductive and the establishment of lactation takes place comparatively independently of the evacuation of the breasts. This twenty-four-hourly fast is helpful rather than injurious to the child, as during this period swallowed amniotic fluid, blood, or vaginal secretions are frequently found in the stomach.

Feeding begins under normal conditions with the commencement of the second day. With regard to the number and time of the feeds, it is advisable to employ from the beginning those rules which are to be followed in the subsequent weeks and months. It is often recommended to feed the infant only three to four times on the second and third days; many children will not drink more often. Others will certainly protest loudly against such fasting. With normal children there is not the least reason for increasing the number of feeds during the newborn period to the usual amount given in infancy as is frequently done. We must emphasize the need that even in the young infant the intervals between the individual feeds should amount to three to four hours, that a nightly interval of six to eight hours should be interpolated, and that the child only be put to the breast five or six times in the course of twenty-four hours. Should six feeds be permitted there should be some special reason for it (smallness of the meals owing to weakness in sucking). Under normal conditions, at any rate, a seventh meal is superfluous.

The observance of a limited number of feeds has various advantages. An increase in their number with breasts yielding milk plentifully involves the risk of overfeeding with all its troubles or other unpleasant consequences. Undoubtedly in the majority of

cases during the first days the total quantities taken in a day with frequent feeds do not exceed, or only slightly, those resulting from infrequent meals, the reason being that at this early period the production of milk is not yet fully established and the child has not yet fully developed its powers of sucking—therefore, when meals are more frequent the amounts taken at each are less; nevertheless, even in these cases it makes a considerable difference to the delicate intestinal mucous membrane of the newborn whether it be affected frequently or seldom by the irritation caused by the food, and whether fresh food reaches the alimentary canal before the previous meal has been passed on to the lower parts of the intestine. It is, however, not only of advantage for the child, but also for the mother when feeds are given as seldom as possible. Nursing during the first days is often associated with considerable discomfort; the tender skin of the nipple is very sensitive, especially when rhagades are present, and these may occur even when the greatest care is taken. Should only one breast be used in each meal, this being recommended when the secretion is normal, then each breast is only made use of two to three times every twenty-four hours. The nightly interval is certainly a great relief for the mother, especially during the first days of confinement, often, it is true, only when the child is taken into another room for the night, since in many cases the long fast is not undergone without crying. The crying of the child can, however, be no reason for deviating from the rules established by long experience as the most useful. The education of the child must begin on the very first day (Czerny). In most cases the infants soon become accustomed to the prescribed arrangement. When this is not the case the children are naturally restless, and of nervous temperament, and will generally cry even when frequently fed.

As has already been said, it is well only to use one breast at each feed, on the one hand to protect the nipples, and on the other to accustom the child to empty the breast so far as possible. Should the child be weak at sucking or the breast yield with difficulty, or be not very rich in secretion, this rule must certainly be broken frequently and both sides be drunk from at each meal; nevertheless, it is advisable always to try and see whether one side is not sufficient. When the feeds are shown to be necessary from both breasts the child must be fed alternately with each breast, so that neither breast may be neglected. Many infants show a remarkable preference for one side, evidently because one nipple fits the mouth better or the breast in question secretes more easily. In such cases it is advisable always to feed the hungry infant first from the breast which is not preferred.

No definite rules can be made with regard to the duration of each meal. In general, it may be said, that during the first days they must be of considerably longer duration than later when lactation is fully established and the child has learnt the technique of sucking. Only in very favourable cases can meals of ten to

fifteen minutes' duration be given to begin with. Often twenty to thirty minutes pass before a meal is ended. Naturally, the child does not drink the whole time. With the first meals a considerable time often passes before the child gets hold of the breast at all properly. Sometimes the sucked-in part of the areola slips out of the mouth and the child sucks at the nipple only without getting any milk. Many infants go to sleep after a few sucking movements or at least stop sucking, with the nipple in the mouth. In such cases the child must be reawakened and encouraged to suck by a few pats on the cheeks or the chin, often also with one or two pats on its buttocks. These little difficulties, which may certainly be considered as normal occurrences, are usually overcome after the first attempt at suckling. Should lactation be established in the meantime, in the majority of cases the meals may be curtailed from the fourth or fifth day. As a rule, the child regulates the duration of the feed itself, as after powerful sucking movements it soaps and falls asleep. It is always easy to decide whether this is due to a feeling of satisfaction or to easy tiring or laziness in drinking. The mother can feel quite easily whether the child has sucked or not, this also being shown by the consistency of the breast. The surest method of finding out is undoubtedly by weighing. Care must be taken that the mother, when she has had to spend a long time in nursing during the first days, does not make the child continue to drink after it has stopped when lactation is established.

Nutritional Requirements and Amounts of Drink

In order to determine the amount of drink necessary for the well-being of the infant, it is best to ascertain the amounts drunk by healthy, normally developed infants and to deduce average values from them. If the numerous statistics, partly obtained from maternity clinics and partly from private practice, be considered, they will cause some reflection. These statistics mostly included only those children who had regained their weight at birth after eight to ten days. This narrow limitation of the conception of physiological development cannot be considered correct in view of our present observations and experience, since there are many infants not corresponding to this claim, but who nevertheless continue to develop normally and must be considered healthy from every point of view. The regular rise in the curve of weight from the day of minimum weight onwards is of much greater importance in determining the conception of "normal" than the regaining of the weight at birth within a given time. The need of nourishment is also certainly related to the absolute body weight of the child at the time. The different individual water contents of lighter and heavier infants and also the different degree of the physiological loss in weight must also be considered. It would perhaps be more useful to bring the nutritional requirements into relation with the minimum

weight (on the third or fourth day of life) rather than with the weight at birth. A very important factor in judging the amounts to be drunk is the difference in the nutritive values of human milk at the commencement of lactation.

The calorie values of human milk amount to:—

| | | |
|----------------------------------|---|-------------|
| According to Rüchser and Heubner | 614.2; 723.0; average value | 656 cal. |
| According to Gaus | 609.0; 740.9; 744.3; average value | 722 " |
| " Schloemann | min. 695.5; max. 876.8; average value | 721.45 cal. |
| " Reyher | min. 754; max. 774; average value | 765 cal. |
| " Engel | calculated average value from the average composition of human milk | 765.05 cal. |

The values obtained from mature milk vary greatly, but the variations at the commencement of lactation and in the colostrum milk must be still greater. The high specific gravity indicates a high concentration. Probably in the majority of cases the upper limit in the above statistics corresponds to the calorie value of the early milk. The lower the amounts of milk taken, probably the higher is the nutritive value of that taken. Reyher thinks that in all cases of low quantitative productivity of the mammary gland compensation takes place in the higher calorie value of the milk.

In this connection the observations of O. and W. Heubner are very instructive; they calculated from one case (by milk analysis) the following calorie values:—

| | |
|----------------|---------------------|
| 4th—7th day | 800 cal. per litre. |
| 8th—22th day | 772 " " |
| 23th—32nd day | 888 " " |
| 33rd—161st day | 830 " " |

The child in question never drank more than 450 gm. milk; its calorie value must therefore have been strikingly high.

Our knowledge of the true calorie value of the first milk is still very incomplete.¹ The hitherto available data as to the chemical composition were based on adequate material for analysis, and also a sufficient productivity of the mammary gland. When there is not enough secretion for analysis it is probable that the small amounts of milk present have a much higher concentration and a correspondingly much higher calorie value.

In order to determine the nutritional demands of a young infant Gaus proceeded in the following manner: two to three hours after

¹ NOTE IN CORRECTION OF PROOFS.—The gap has been filled by calorimetric investigations by Langens, Rott and Edelstein ("Der Nährwert des Kolostrums," *Zeitschr. für Kinderheilkunde*, 7, 1913, 216). They showed that the nutritive value of the mammary secretion is often very high immediately after birth and may reach twice the value for mature milk, and the calorie value sinks gradually at first during about five days, when it reaches the normal calorie value of human milk. The values certainly vary within wide limits (200—1,200 cal.); in the yellow, viscous, sticky milk they are high, and in the thin, watery, early milk relatively low.

the last feed a quantity of milk was removed from one of both breasts by means of a pump equivalent to the amount usually taken by the child in question; the milk so obtained was investigated with a calorimeter and its caloric value determined. It appeared that the adoption of an average value of 722 calories per litre from three varieties of milk in the first ten days often results in a physiological growth with an energy quotient of 50 calories or less. In 100 cases of breast-fed infants the lowest energy quotient associated with regular increase in weight did not in any case amount to more than 44 calories until the eleventh day. Heubner and Langstein came to the same conclusion that in the first week the child may thrive on an average supply of 50 calories; according to Beutliner, the average energy quotient amounts in the first week to 50 calories.

Gaus, however, rightly points out that beside the energy quotient the water content of the food given is also of the greatest importance. "Undoubtedly during the first few days of growth infants do not only derive their increase in weight from the caloric value of the food, i.e., from the solid part, but also from the water." Probably the "growth" in the first week of life is chiefly such that the water lost during the first few days is replaced, and also partly involves the restitution of the excreted solid constituents; only when the weight at birth has been almost regained, that is after complete restitution, does a true deposit of body substance begin.

Besides the nutritive value of the food the volume taken is also important in determining the nutritional demands, this being particularly the case in the first week with its great variations in the water-content of the organism.

In this connection an observation of Cramer regarding an artificially fed child forms a very instructive example. The child received one-third milk with the customary addition of lactose.

| | | | | | | | | | | |
|------------------------|------|------|------|------|------|------|------|------|------|------|
| Day of life | 1st | 2nd | 3rd | 4th | 5th | 6th | 7th | 8th | 9th | 10th |
| Weight | 3140 | 3070 | 2670 | 2890 | 2940 | 3000 | 3050 | 3120 | 3160 | 3170 |
| 24-hourly amount drunk | 15 | 30 | 45 | 100 | 170 | 220 | 260 | 290 | 350 | — |
| Energy quotient | — | — | — | 14 | 25 | 29 | 35 | 37 | 44 | — |

Considering the caloric value of one-third milk to be on the average 400 cal. per litre it appears that from the day of minimum weight until the tenth day the child was on a diet of gradually increasing energy quotient of 14 to 44 cal., its weight at birth being regained on the ninth day; its growth was therefore normal in the strictest sense of the word. Cramer therefore considers that the degree of dilution of the milk is of less importance than the absolute amount of nutrition.

It follows from the above that the energy quotient of 50 to 100 cal. requisite for the age of infancy is not necessary for the normal development of the infant for the first 8 to 10 days, but that a diet yielding 50 cal. per kg. body weight is quite sufficient. In any case it is possible during the first days of suckling for the

energy quotient to be considerably below this figure, provided that the volume of the food is up to a certain limit.

Cramer considers that the best method of feeding is one in which the greatest increase in weight follows the smallest amounts of food. In his opinion, there exists a physiological, proportional optimum between supply of nourishment and increase in growth. Cramer describes the relation between the increase in weight (from minimum weight to the weight on the tenth day) and the amounts of food used from the first to the tenth day (without considering its nutritive value) as the "quotient of nutrition" (*Nährquotient*). In a breast-fed infant which developed normally

it amounted to $\frac{260}{1520} = 17\frac{1}{2}$ per cent.

With overfed infants the nutrition quotient is lower, with underfed, higher; in the newborn child according to Gauss, Cramer's nutrition quotient amounts on the average to 10. It appears that the size of the nutrition quotient is dependent on the body weight, in so far as that an infant with a nutrition quotient < 20 always exceeds 3,200 gm. weight.

On purely practical grounds we are interested in the physiological nutritive demands of newborn children for two reasons: (1) In order to determine whether a child is adequately fed or over or underfed; (2) If when the nutritive demands are covered the development of the child is physiological and if when we see variations from the normal course (with regard to weight, outward appearance, &c.) we can diagnose under- or overfeeding or a pathological process in the organism, in other words, whether the abnormality is on the part of the child or of the amount of food taken.

If one wishes to decide whether the amounts of nourishment drunk during the first days of life, determined by weighing, adequately meet the physiological needs, it will be found that not much progress is made with the calculation of the energy supplied owing to the reasons already given; in individual cases one does not know which caloric value to calculate from. It is better to proceed volumetrically and to compare the figures obtained in special cases with the numbers determined empirically by measuring the amounts of drink taken by normal healthy breast-fed infants. Certainly only an approximate estimate of the actual nutritive demands is obtained in this way, but the figures enable one to obtain a good idea of the fluid requirements—and this appears to be of greater importance during the first days of life.

Finkelstein recommends the following formula for the orientation of the requisite amounts of drink during the first days: (day of life - 1) \times 70 to 80 gm.; thus for the fifth day of life: 4×70 to 80 gm. = 280 to 320 gm.

In the tables shown below (pp. 106, 107) are some of the observations, now available and already fairly plentiful, concerning the

AMOUNT OF DRINK OF HEALTHY FULL-TERM

| Author | Weight at Birth | DAY OF | | | | | | | |
|-------------------------------------|-------------------------|--------|-------|--------|--------|--------|--------|--------|--------|
| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| Krüger (1875) ... | — | 12-15 | 26 | 192 | 234 | 363 | 441 | 501 | 528 |
| Reising (1893) | 9060 (2200-3650) | 35.3 | 100.8 | 170.6 | 220 | 271.5 | 296.6 | 297 | 333 |
| W. Cazemajou (after Klemm) | 3126 | 17 | 94 | 193 | 300 | 352 | 381 | 462 | — |
| Denecke (1886) (after Jäschke) | — | 44 | 135 | 192 | 256 | 352 | 385 | 381 | 411 |
| Tamm and Haer also after Jäschke | — | 26 | 75 | 88 | 155 | 218 | 233 | — | — |
| Cranner (1901) ... (after Klemm) | — | 1.5 | 16.83 | 50.42 | 102.06 | 226.66 | 346.66 | 311.66 | — |
| Perr (1902) ... | 3528 | 4 | 90 | 177 | 313 | 436 | 549 | 541 | 597 |
| Glas ... | — | — | 52.0 | 140.84 | 235.46 | 298.84 | 335.92 | 367.64 | — |
| Amestrucci (1903) | 3493 | — | 22.5 | 20.9 | 175.5 | 217.6 | 247.49 | 281.5 | — |
| Bestheuer (1902) | — | 17 | 91 | 190 | 302 | 348 | 381 | 450 | 476 |
| Klemm (1907) ... | 3091 | — | 15 | 100 | 170 | 460 | 449 | 483 | — |
| Jäschke (1909) ... | 2700-3416 | 19 | 90 | 193.7 | 260.4 | 339.3 | 402 | 415.6 | 420.5 |
| Opitz (1911) ... | 3000-3500 | 37.7 | 107.8 | 206.6 | 371.5 | 414.6 | 462.8 | 455.3 | 485.7 |
| Jäschke (1912) | 2200-2500 | 45 | 65 | 111 | 173 | 184 | 272.6 | 293.9 | 317.4 |
| | 2500-2900 | 36.2 | 95.4 | 126.4 | 239.4 | 255.7 | 275.8 | 308.6 | 316.9 |
| | 2900-3300 | 34.0 | 87.5 | 132.14 | 228.1 | 255.22 | 319.20 | 320.23 | 349.31 |
| | 3300-3700 | 23.6 | 91.8 | 218.8 | 341.1 | 435.11 | 444.12 | 497.2 | 461.12 |
| | 3700-4000 and higher | 20.7 | 97.7 | 223.7 | 358 | 424.10 | 454.6 | 506.1 | 467.9 |
| Author's observations | 2500-3000 | — | 54 | 173 | 263 | 377 | 354 | 362 | 390 |

BREAST-FED CHILDREN. AVERAGE FIGURES.

| L.I.B.R. | | | | | | | Final weight (day in brackets) | Number of daily meals | Remarks |
|----------|-------|-------|-------|-----|-----|--|--------------------------------|-----------------------|---|
| 9 | 10 | 11 | 12 | 13 | 1 | 4 | | | |
| 621 | 648 | 701 | — | — | — | — | — | 6-9 | 2nd day 6) 3rd-5th " 8) Meals 6th-11th " 9) Average of 20 cases |
| — | — | — | — | — | — | — | — | — | " 6 " |
| — | — | — | — | — | — | 3024 (7) | — | — | " 11 " |
| — | — | — | — | — | — | — | — | — | " 16 " |
| — | — | — | — | — | — | — | — | — | — |
| — | — | — | — | — | — | — | — | — | Average of 5 cases (amongst them differences in weight of 1,050-4,360) |
| 592 | 603 | 377 | 391 | 390 | 600 | 3404 (7) 3580 (14) | — | — | Average of 7 cases (robust children) |
| — | — | — | — | — | — | — | — | — | — |
| — | — | — | — | — | — | 3333 (7) | — | — | Average of 10 cases (weight 2080-4520) |
| — | — | — | — | — | — | — | — | — | Average of 16 cases (also of other authors) |
| — | — | — | — | — | — | 3096 (8) | — | 7 (after 5th day) | For the first 4 days less frequent meals. Average of 3 cases (in a single family) |
| — | — | — | — | — | — | — | — | — | Average of 18 cases. Average values, but no minimal values |
| 467.6 | — | — | — | — | — | Weight at birth regained within 9 days | — | — | Average of 75 cases |
| 359.3 | 395.2 | — | — | — | — | — | — | 4-6 | — |
| 364.7 | 313.7 | — | — | — | — | — | — | | — |
| 388.20 | 418.2 | 355.5 | 413.7 | — | — | — | — | | — |
| 484.1 | 482.8 | 431.0 | 430.6 | — | — | — | — | | — |
| 484.1 | 482.8 | 498.2 | 468.3 | — | — | — | — | | — |
| 341.9 | 547.7 | — | — | — | — | — | — | — | — |
| — | — | — | — | — | — | — | — | 5-6 | Average of 25 cases |

twenty-four-hourly amounts of drink required by healthy breast-fed infants during the first two weeks of life.

As may be seen, the average values obtained for the "normal" infant vary within very wide limits. Still greater differences are found when the results of individual observations are compared, as may be found in contributions by Nordheim, Selter, Weichart, White and others, besides in the writings of the authors quoted in the table.

The size of the individual meals is also subject to great variations during the first week. In tests it is therefore advisable always to determine the amount of several meals since no conclusions as to the daily amount drunk can be drawn from the size of one meal. Frequently the first meal in the morning, after the nightly interval, is the largest, but this is by no means invariable. A typical curve as Engel has found regarding the size of the meals in later infancy (maximum in morning, fall during the middle of the day and a further rise in the evening) cannot be recognized during the first few days of suckling. The first feeds are, even when the child makes sucking movements, frequently so small that they cannot be measured by weighing, none the less some children drink weighable amounts even on the first attempt; usually the amounts vary between 5 to 20 gm.; 20 gm. is but rarely exceeded on the first day of suckling. The low amounts may also persist during the second day of lactation (= 3rd day of life), especially when lactation is not yet established or the breast yields with difficulty. When once the true secretion has begun the meals soon increase in size (20 to 60 gm.) and now under normal conditions a continuous rise in the amount taken at each meal follows, the amounts reaching 80 to 100 gm. and more; large amounts always remain isolated however and alternate with much smaller amounts. The variations in the size of the individual meals may be seen in the accompanying curves (figs. 14 to 21).

For practical purposes the principles regarding suckling during the first days may be summed up as follows; the child who receives nothing or at most only small amounts of weak tea sweetened with saccharin on the first day, is put to the breast for the first time twenty-four hours p.p. If the case be not one of a small premature or markedly debilitated child, for which particular rules are applicable, the attempt will be made from the first day of suckling onwards to feed the child according to the usual plan: five meals in twenty-four hours, alternating between the right and left breast, at three- to four-hourly intervals with a longer nightly interval interposed during which the child receives nothing under the best conditions, but if very restless it is given relatively small amounts of indifferent liquids. The meal times are best arranged as follows:—

- 5 to 6 a.m. (as soon as the child wakes and cries).
- 9 to 10 a.m.
- 1 to 2 p.m.
- 5 to 6 p.m.
- 9 to 10 p.m.



FIG. 16.—Age 19. II para, who nursed her child for over a year. ♀. Very favourable suckling conditions. Minimum weight already reached by the second day of life. Ideal curve.



FIG. 17.—Age 25. II para. First child fed only 14 days. ♀. Small child. On the third day temporary feeding with maternal milk initiated artificially on account of hardening of the breasts. Quantity of feed not very plentiful, but not abnormally low.



FIG. 18.—Age 29, 1 para. 2. Favourable suckling conditions. Slight decrease in weight, but lack of the normal increase in weight, although the foods were plentiful in amount. Fervent vomiting.



FIG. 19.—Age 29, IV para. (The earlier children breast-fed for one week only.) Breasts very rich in milk, and a large amount taken at each feed. Slight decrease and rapid regaining of weight at last. Violent irritation of breasts. The child ought only to have had five meals; also the tea given to the child on account of restlessness during the night was superfluous. ("Ideal curve," with a by no wise "ideal" clinical course.)



FIG. 20.—Age 10. 1 para. ♂. Normal weight course and normal feces. Transitory fever on second and third day.

A. impenabilis is measured.



FIG. 21.—Age 21. II para. ♂. Comparatively marked decrease in weight, but after minimum weight constant increase. Transitory fever from second to fourth day. Favourable reeking conditions. Bevel irritation.

The duration of a meal is on the average eighteen to twenty minutes, in particularly favourable cases still less; if the child has a long pause during the meal or has to be encouraged to drink a meal may last half an hour or more.

If it appears that after two or three days a child does not drink enough with five meals and after reaching the minimum weight does not increase or even loses weight, then a sixth meal must be fitted in. The meal times are then somewhat as follows:

5 to 6 a.m.

9 a.m.

12 to 1 p.m.

4 p.m.

7 p.m.

10 to 11 p.m.

If the cause of the small amounts of drink taken lie not so much in the fact that the child sucks weakly or refuses larger amounts, as in the originally low production of the breast or of the difficulty of its yield, then the five meals a day routine may be continued but both breasts used each time, or the milk not taken by the child may be pumped off and given to the child immediately after it is taken away from the breast. The last-mentioned method is much to be recommended; the child is kept on its regular meals and the breasts are thoroughly emptied. Often after one or two days of additional feeding the child will drink the requisite amount without hesitation directly from the breast. If during the establishment of lactation the breast does not yield enough milk, tea or water may be given after each feed.

The determination as to whether the child is obtaining enough nourishment is made from the general appearance and behaviour of the child, and from the quality of its motions; the most valuable information is naturally given by the determination of the amounts drunk and the estimation of the progress of the weight by daily weighings.

The introduction of a seventh meal, as also the after-feeding with milk or a neutral fluid after the physiological beginning of lactation (i.e., after the fourth day), only proves to be necessary under rare circumstances which strictly speaking can no longer be considered normal.

Additional feeding with fluids is quite unnecessary as a rule with strong infants and favourable suckling conditions. Should the child show itself to be unskilful in obtaining milk at the first attempt, should the breast clog with difficulty, the establishment of lactation be delayed, the child be restless, then additional feeding with fluids is indicated to prevent further desiccation. The meal times should still be adhered to, the insufficient breast feeds being supplemented; no fluid must be given during the intervals. During the night even when the meals are adequate additional fluid may be given if the child be very restless. There is nothing to be gained

by being too strict on this point. The dummy may also be used with very restless infants since this is quite harmless if kept aseptic; if this be unsuccessful a meal of tea or water may be conceded. When from excessive theoretical zeal everything is forbidden, the disturbed mother will often disobey the doctor and put the child surreptitiously to the breast during the night. We do not mean to say that one should not strive to keep the nightly interval so far as possible. The child itself usually decides the amount of supplementary fluid; 20 to 30 c.cm. are usually sufficient during the first nights. It does not matter much whether water or tea be given or whether feeding take place from a bottle or with a spoon.

(B) DIFFICULTIES IN NURSING

All doctors who have studied the diseases of infancy agree in strongly recommending natural feeding; that is, in the majority of cases, suckling by the mother herself. It must at the same time be borne in mind that natural feeding even by the most willing mothers is often associated with very considerable difficulties of various natures. Since the therapeutic measures for the alleviation of the difficulties of suckling are different according to the origin of the trouble, it is of the greatest importance for every doctor who directs the feeding of an infant to be able to recognize and distinguish between the various difficulties in suckling which arise at the beginning of lactation. They may originate from the child or from the mother.

1.—Difficulties in Lactation on the Part of the Child

(A) WEAKNESS IN SUCKING.

Just as premature infants are often unable to suck at the breast, there are also full term infants who are decidedly weak or lazy at suckling. Such children do not get hold of the nipple or they let it go after a few weak sucking movements, they then either suck a little more without drawing any milk or fall asleep. These symptoms, not always easy to distinguish whether due to weakness or small requirements of food, often disappear after the first or second day of suckling, in the most favourable cases after the first attempt; but sometimes, in spite of every care, days, even the whole of the first week, may pass without the child's learning to suck successfully.

The question here concerns, not always, though relatively often, small delicate newborn children of low weight at birth, a class of infant for which no current term is in use. They are neither of defective vitality nor of anomalous constitution, they show themselves to be fully developed according to the demands of extra-uterine life, they increase in weight possibly somewhat more slowly though regularly, they may regain their birth weight later than usual and

throughout the first months give the impression of being delicate, in the course of the second half year however they make up for their deficit in weight and subsequently develop into perfectly normal children. The entire condition generally reminds one of a healthy prematurely born infant, and in fact many such infants may be undiagnosed premature births, though this is certainly not always the case. To such infants who show definitely the characteristics of premature children, although they are undoubtedly born after the normal termination of pregnancy, must be attributed a functionally abnormal backwardness (see also p. 146).

If such a child does not suck at all or drinks very minute amounts incapable of being weighed then the only correct way of feeding is to use a bottle or a spoon containing the mother's milk previously pumped or squeezed out. Thereby, not only is the secretion of the mammary glands maintained but underfeeding is avoided as far as possible. A certain amount of underfeeding not only may, but should exist, in order to keep the child's appetite keen. The child should be put to the breast regularly, or the attempt should be made; it is then fed with the milk drawn off in small measured doses, even if the supply is plentiful. In the majority of cases the infants begin to suck after a few days especially when the secretion is well maintained and the milk can more easily be sucked out. Such cases are more difficult when the breast contains none or only a small amount of milk to be drawn off, that is, when a difficulty of suckling also exists on the part of the mother (*vide infra*).

When the child takes the breast but drinks so small an amount that underfeeding results it is also best to give it pumped off milk immediately after the feed, thus completing the feeds without adding to their number.

An increase in the number of feeds only appears to be indicated when the child refuses further nourishment at the end of the breast feed. With this class of poorly sucking infants (including the relatively poorly sucking, who should be able to drink from a breast yielding with difficulty but not sufficient) it is often possible to avoid continued underfeeding by more frequent feeding (seven to eight times in twenty-four hours) causing a considerable rise in the total amount drunk (Rietschel). It would certainly be wrong in such cases to adhere to the strict method of five feeds a day. The technique of feeding in these cases is much the same as that generally employed for premature infants.

When possible, i.e., when the child does not refuse a larger quantity of food, it is advisable to feed after each meal with pumped off milk, maintaining the infrequent meals. This is advantageous both for the child and for the mother during the first few days of suckling so that the sensitive skin of the nipple is saved so far as possible. Moreover, suckling to begin with, even without rhagades being present, is often quite painful and fatiguing and exhausting for the mother. If it appears that a child is weak at sucking and

does not obtain enough in five meals, then six may be permitted without hesitation. The addition of a seventh or eighth meal is not, as a rule, needed until the end of the first week.

(B) BREAST-SHY INFANTS.

In rarer cases a peculiar disinclination of the child to suckling at the mother's breast may interfere with nourishment, without the child giving the impression that it is weak at suckling or without appetite and without there being any fault to find with the mother's breast. Every time the child is put to the breast it begins to cry, turns its head obstinately to one side, pulls at the nipple or shies at it, in short, it is impossible to make the child suck quietly.

These "breast-shy" infants usually drink quite well if food be given them in a bottle (Schlossmann). In such cases it would naturally be a mistake always to satisfy the needs of the infant in such a way. The children can only be compelled to suck through hunger so that suckling can only be carried out when a certain degree of underfeeding is present. In such cases it is advisable to keep to intervals of three to four hours between the meals. In order to overcome the unwillingness of the infant Schütz advises the following procedure: the erect nipple is put into the child's mouth and at the same time a stream of milk is squirted into it. At this early stage the sense of taste is procured by a relatively much wider area of mucous membrane than in the adult, the tongue, the hard and soft palate, the mucous membrane of the cheeks, the posterior pharyngeal wall, &c., all being sensitive. By moistening these parts with milk the appetite may often be stimulated and the child induced to continue suckling. After the attempts at suckling, which demand much patience and endurance on the part of the mother and skill and encouraging advice on the part of the doctor and of the attendants, the infant must be given principally fluids (water and tea), but not too much milk, even if the amount forthcoming from the mother's breast is plentiful. Should the "breast-shyness" of the child be caused or increased by nipples which cannot easily be held or by a breast yielding with difficulty these defects must naturally be dealt with in the first place and removed or reduced as much as possible. In uncomplicated cases the prognosis of suckling is favourable if the treatment be correct, even if suckling is not established before the second week.

Needless to say the mouth of the child should also be inspected in case there is any painful inflammation (stomatitis or epithelial defect) which might interfere with suckling. The importance of rhinitis as a hindrance to suckling must not be under-estimated, for this greatly limits or entirely prevents inspiration during suckling and thus compels the child to breathe through the mouth. The possibility of the rare congenital absence of the sucking reflex must also be borne in mind; such infants will also not suck at a bottle.

A very peculiar case was described by Osi: a healthy newborn child was unable to drink because every sucking movement was absent. Osi, by the

use of a very long nipple shield was the child able to drink. Since no anatomical cause for the disability was present, it was supposed that the starting point of the sucking reflex was placed unusually far back on the base of the tongue.

The so-called "tongue-tie" (shortness of the *frænum linguae* at its insertion into the apex of the tongue) cannot interfere with suckling; this follows direct from the mechanism of the art of suckling.

2—Difficulties of Suckling on the part of the Mother

(A) BAD DEVELOPMENT OF THE NIPPLE, UNSUITABLE SHAPE OF THE BREAST.

Badly developed nipples may constitute a hindrance to suckling which often leads to a very marked degree of underfeeding and is not infrequently given as a cause of incapability for nursing. They are particularly troublesome when associated with under-development of the glandular parenchyma and a corresponding smallness of the secretion.

Kehrer divides malformations of the nipples into the following classes:—

- (1) Smallness of the nipples, *microthelia*.
- (2) Fissured nipples, *mammilla fissi* (split running transversely across the apex, dividing the nipple into an upper and a lower lobe).
- (3) Nodulated nipples, *papilla verrucosa*.
- (4) Concave or depressed nipples, *papilla circumvallata*. The nipple lies deeper than the surrounding parts of the areola. The areola surrounds the otherwise well developed nipple as a wide ring (*papilla circumvallata aperta*) or the nipple is shaped like a flattened cone with depressed apex. Only on close inspection can it be seen that the covering of the cone is formed by the thickened areola and that at the bottom of the depression a little nipple is hidden (*papilla circumvallata obtecta*).

Basch distinguishes only three kinds of deformity, the *papilla plana*, *fossa* and *invertita* (= 4b of Kehrer's classification). Etiologically he considers the deformities as disturbances of development which take place from a standstill in the various stages of growth passed through in normal development. The most primitive stage of development is that of the concave nipple which really exhibits on an enlarged scale the condition typical of the normal newborn child. In this condition the nipple is always atrophied. Should a circular constriction of the areolar musculature take place over the papilla the circumvallate condition results. More advanced stages of development correspond to the *papilla fissi* and *plana*.

Stuhl not only distinguishes the flat and depressed nipple but also the infantile or pointed nipple. While the first two may be held with difficulty the pointed nipple is too small and easily slips out of the child's mouth.

With a breast which secretes relatively well it is nearly always

possible to rectify a nipple which at first appears unsuitable for the child that sucks, not only at the nipple, but also, if it does it properly, at part of the areola. The knowledge required for these cases can only be learned from experience. It is not overstating the case to say that the success of suckling depends in the first place on the skill and experience of the nurse assisting the mother.

The inability of the child to hold the nipple often depends less on the shape of the nipple than on that of the breast itself. Thus the child is often unable to drink easily from the turgid breast of a young primipara even though the nipples be well formed, because, as in the case of the breast hardened by commencing lactation, it is not able to get a good hold on the areola. In other cases difficulties arise when a small infant has to suck from a large, very broad spongy breast (Jaschke). As Basch has pointed out there is a definite inverse proportion between the height of the nipple and the wideness of the areola, the higher the nipple, the smaller, as a rule, the areola and vice versa.

It is often advised that care should be taken even before pregnancy to develop the nipple well. Whether an interference with its development is to be feared from too tight clothes or corsets (Walcher) is, according to the above conception of Basch, at all events doubtful. He is of opinion that care must be taken as early as the first months of life to see that the development of the nipple is not interfered with by a particularly hard horny layer of skin, and he therefore recommends the application of substances dissolving keratin (compresses with 1 to 2 per cent. solution of caustic soda). It is certain that such far-seeing prophylactic methods, which see in the infant the potential nursing mother, will be but rarely put into practice. Usually attention is only directed to the fully formed nipple on the occasion of lactation or during pregnancy. The frequent application of the breast pump in such cases is advisable, not only because it increases the secretion of milk, but also because the nipples are pulled out by the suction of the pump. This may also be done by the application of a small ball pump a few minutes before the meal (fig. 23). With nipples which are very difficult to hold the nipple shield often, undoubtedly, does good service. If the child possesses a sufficient power of suction, and if the breast contains a sufficient amount of easily removable milk, then the results of indirect application are often very satisfactory, although this method cannot be recommended as the usual method of feeding; the child obtains food, and the nipple is drawn out by this combined method.

Preferable to the usual nipple shields, the glass rims of which compress the lactiferous ducts and may thus reduce the secretion, is that of Soern, which is made entirely of rubber known under the name of "Infantibus." This is ingeniously fixed to the breast by vacuum (fig. 22).

In order to raise up a depressed nipple Kehler recommends operative pro-

cedence. Hesch carries out a subcutaneous resection of the areolar muscle and follows it up with orthoplastic treatment (continuous pulling out of the nipple to bring it into the condition of a papilla plana). The most suitable time for the operation is during girlhood or during the first months of pregnancy.

(B) RHAGADES OF THE NIPPLE.

Rhagades and fissures of the nipple form a very important hindrance to suckling, and in their treatment great difficulties are often met with. This condition occurs very readily after the first attempts at suckling in a great many women. They are found most frequently in primiparae with tender nipple skin; after the second



FIG. 22.—Right breast: Usual nipple shield. Left breast: "Infantilon."



FIG. 23.—Right breast: Bell pump. Left breast: Aspirating nipple shield.

child, and in multiparae who have already suckled, they occur less often. The latter are, however, not exempt, since there are women who get sore nipples at the beginning of every lactation. The lesions consist partly of linear bleeding fissures, partly in more superficial erosions, and defects in the outer layers of the epidermis; these coxe and finally granulate. The rhagades are situated either on the apex or on the base of the nipple and are often radiate. The escaped blood dries during the intervals between the feeds, forming brown scabs, which are removed when the child sucks, so that bleeding begins again. The hemorrhage is sometimes quite considerable, this being shown by the fact that melena spuria resulting from sucking from wounded nipples may under certain circumstances be mistaken for melena vera.

We are not in possession of a certain prophylactic method against the occurrence of rhagades; they occur in spite of all treatment with a view to hardening the nipple during pregnancy (see p. 96).

The difficulty in treating rhagades is due to the fact that it is necessary to permit the injurious agent which has caused the injury to continue its action, since this agent is the sucking child. Treatment must therefore be directed chiefly towards protecting the nipple from the sucking of the child. This may be done by using a nipple shield and thus giving protection from the direct pressure of the child's jaw. It is true that the glass rim of the usual nipple shield rubs against the rhagades when they are situated laterally. In such cases Stern's nipple shield is very useful; the sucking portion which covers the nipple is sufficiently large to protect it from pressure and rubbing to a considerable extent. The healing process is certainly disturbed less in this way. It must not be thought that the healing of the fissures is prevented by the direct sucking of the child; it is really only delayed. In courageous women who bear the pain and allow the child to continue to suck complete healing is seen after a time even after widespread rhagades. It may be said that a favourable prognosis is almost always justified; only the very painful acute condition must be overcome. The fear that infection of the gland may take place from rhagades and result in mastitis is quite unfounded in the great majority of cases if the breast be kept clean. It cannot, however, be avoided with absolute certainty though. From this point of view the use of a nipple shield is also justified as a prophylactic measure. "Disinfection" of the child's mouth is impossible, and any attempt to carry it out had better be prevented in the interest of the child.

Should the pain be unbearable both on direct and indirect suckling, it may sometimes prove necessary not to use the affected breast for a few meals or even for a few days. If only one nipple be affected, suckling may take place from the opposite one, but in the majority of cases this is also affected before long. The partial leaving off of suckling also has its disadvantages. The under-feeding of the child arising in this way is not so dangerous as to make it advisable to compel a woman to continue suckling in spite of the pain, which may be very great; it is best to carry on with indifferent fluids and pumped-off milk so far as the latter may be available. Far more unpleasant in breasts rich in milk is the engorgement which takes place when suckling ceases; this causes the woman further pain and also delays the healing of the rhagades. In such cases provision ought to be made for the discharge of the glands. It entirely depends upon the sensitiveness of the woman at the time whether this is to be done by putting the child to the breast or by pumping off the milk. The latter process is sometimes a very painful one, especially if the sore places are situated laterally, and great care must be taken as occasionally the rhagades cause considerable hæmorrhage. If no signs of mammary engorgement occur, a good result is usually obtained by stopping regular suckling

for a time and putting the child to the breast only now and again. The fear that this might cause lactation to cease is quite unfounded. However, the ordinary five-meals method, in which each breast is only used two to three times a day, is in itself a very conservative procedure.

It is only in very nervous, sensitive women, and especially in those unwilling to suckle, that rhagades constitute a contra-indication to suckling, though it should only be considered an absolute bar in extreme cases. Judicious advice is often very beneficial; it must always be borne in mind that the condition is very troublesome but transitory and not dangerous.

The methods of treatment of rhagades are very numerous. The treatment with ointments enjoys the greatest popularity. A simple boracic ointment generally answers the purpose (3 per cent. boracic vaseline-lanoline); this is best applied in a thick layer spread on a piece of lint and laid on the sore nipple. Before suckling the ointment is removed with a little oil or dilute alcohol (50 per cent.), this being washed off with boracic lotion. The wound may be treated with caustics before the application of the ointment, but not in pencil form, since this causes considerable scabbing, after the removal of which the surface of the wound is often greater than before; a 5 to 10 per cent. solution must be used. A dilute (1 per cent.) caustic ointment with addition of 10 per cent. balsam of Peru may be applied. Instead of silver nitrate, tannin alcohol may be applied with a brush several times a day (Engel).

B. Acid, boric, 20—50
Glycerine, 200
Sgt. var. rect. ad 1000.

Treatment with silver nitrate also alleviates the pain. For the same purpose a 10 per cent. anæsthesin ointment is recommended. Schiller advises the following ointment for causing relatively rapid healing of the skin.

| | | |
|-------------|-----|-----|
| Acid, boric | ... | 50 |
| Zinc, oxid. | | 100 |
| Naphthalan | | |
| Alip. lan. | ad | 150 |

The ointment should be applied after the wound has been opened as widely as possible, the nipple should be cleaned with oil before each meal, some milk should be pressed out and then the child applied to the breast. Scarlet-red ointment has a similar action. The application of giro ointment (2 per cent.) is also well spoken of. A water soluble preparation known by the trade name of "Etoga" forms an excellent ointment basis, because it penetrates well into the tissues and can easily be removed with aqueous solutions before the child is put to the breast (Borebaga, Apisbaga, &c.).

Of less use is the dry treatment (e.g., with orthoform dusting powder) or the application of wet compresses, e.g., with compresses soaked in boracic lotion, which are covered with waterproof material and cotton wool and bandaged on. Marfan recommends the anti-

septic and astringent benzoic acid, which he prescribes in the following way: A pad soaked in the following solution is applied to the sore nipple.

| | |
|-----------------|------|
| Aq. iostearum | 40°0 |
| Glycerine | 20°0 |
| Borax | 5°0 |
| Tinct. benzoin. | 12°0 |

It is then covered with waterproof material and bandaged on. As a prophylactic Fischl recommends tannin glycerine in 5 to 10 per cent. solution.

(c) DIFFICULTIES IN SUCKLING DURING THE ESTABLISHMENT OF LACTATION, WITH MASTITIS AND WITH POORLY SECRETING BREASTS.

Both the hardening of the breast at the time of the establishment of lactation and also the difficulty of evacuating poorly secreting breasts belong in general to physiology and have already been dealt with. Thus the hardening of the breast only gives rise to difficulties in suckling when it lasts a long time and is very excessively developed. The breasts sometimes become as hard as a board and remain in this condition several days; in attempting to suck the child slips away from the turgid breasts, obtaining hardly anything to drink, although in such cases the milk often flows spontaneously and may soak the body and the bed clothes. The mother and child are best helped by pumping off the milk, the former because this diminishes the painful tension even if only small amounts can be removed. The child is benefited because it can be fed on the evacuated milk. In such cases, however, very unpleasant situations may arise; the mother suffers great pain, both evacuation by the pump and application of the child are very painful (combination with rhagades is very frequent), the amounts of milk evacuated are not infrequently so small that they do not satisfy the child and it becomes restless. Fortunately it is always a transitory condition which has at most but one danger, that of being the cause of mastitis due to engorgement. Usually, however, the secretion begins after a few days and the painful tension subsides. The prospect is usually very bright and the prognosis of suckling favourable. At the time of the establishment of lactation the child should be given enough indifferent fluids to drink.

Schiller is of opinion that the mastitis, excluding a few rare cases, commences as a mastitis due to engorgement and not as the phlegmonous form caused by rhagades. He believes that the disease arises owing to the infection of the obstructed milk with bacteria, which usually grow in the excretory ducts. This is not the place to enter more closely into the etiology and treatment of mastitis; as a rule, usually the child has passed through the newborn period before mastitis becomes fully developed in the mother. None the less its commencement often starts in the first week. At this time, suppuration of the inflamed parts may possibly be avoided,

by the early application of Bier's hyperemic treatment; the gland is congested daily once or twice every three-quarters of an hour and subsequently evacuated of milk as far as possible, especially from the congested acinus; this may be done manually by careful centripetal massage or by the milk pump. The breast pump is always used when the healthy breast has just been emptied, at the following meal the child is applied to the affected side. In the interval a dressing with 50 per cent. alcohol or aluminium acetate is put on the breast and the latter lightly suspended by a bandage.

Mastitis, certainly in its early stages, is no reason for not allowing the child to nurse. The evacuation of the breast is advantageous for the mother and does not harm the child. According to Schlossmann suckling may be continued until the mastitis is ready to be incised, provided that the milk is free from leucocytes. If the inflammation is so great that suckling causes unendurable pain or if suppuration takes place, one is compelled to let the child drink only from the healthy side. Since the secretion is usually well established at this time and since mastitis as a rule only affects breasts which secrete plentifully, the milk of the unaffected side usually suffices or only small amounts of additional nourishment are required. After incision suckling may and should be recommended as soon as possible.

The poorly secreting breast leads to difficulties of suckling when there is a disproportion between the ease of yield of the breast and the sucking power of the infant. If sufficient milk can be obtained with the pump or by pressure the same method is resorted to as in the case of true weakness of sucking, that of subsequent feeding with drawn off mother's milk. When, in spite of apparently good filling of the breast, sufficient milk can neither be obtained by the child nor by the pump nor by manual pressure, then a form of milk shortage is present which is very akin to a true hypogalactia. These conditions tend to improve with time, as soon as the glandular secretion is more plentiful and the sucking power of the child a little stronger, but at the commencement of lactation the difficulties are often very considerable.

(D) ²HYPOGALACTIA. THE MEANING OF "CAPABILITY TO SUCKLE." DANGERS OF UNDERFEEDING.

If in spite of the absence of the above hindrances, suckling does not begin about the normal time, it may be assumed that the most unpleasant type of obstruction is present, both for mother, child and doctor, *viz.*, hypogalactia. That there are breasts deficient in glandular substance is shown not only by their deficient secretion but also by anatomical conditions. Together with the type of breast which is rich in parenchyma there is a type characterized by plentiful connective tissue in which the specific glandular parenchyma is poorly developed in spite of the full development of the fibrous corpus mammae (Engel).

The diagnosis as to whether a breast is good or bad, whether it is only poorly secreting but rich in parenchyma, or whether it is a question of a breast poor in milk, is difficult to decide at the time of commencing lactation. Certain conjectures as to the quality of the gland may be made from the presence of a plentiful venous plexus in the skin, which indicates an abundant blood supply and a satisfactory secretion, also from the presence of radially arranged striae (due to stretching) which indicate that an increase in the glandular substance has taken place. Breasts poor in parenchyma certainly often have a soft, downy or a regularly solid consistency, but neither the size of the organ nor the results of palpation justify definite conclusions on this point. There are very big, expansive breasts, the large mass of which is chiefly made up of fat and connective tissue, and on the contrary small flat breasts which hardly protrude from the thorax at all but which are none the less relatively rich in parenchyma and give excellent results in suckling. In thin women, especially at the time of commencing lactation the filling parts of the gland often feel to the touch like a cord-like reticulum; with large spongy breasts which are flabby to the touch, it is, however, often practically impossible by palpation to distinguish connective tissue and fat from glandular substance. The amount of fluid that may be expressed does not always yield definite conclusions. Small breasts out of which milk can only be expressed a drop at a time with trouble, often yield relatively large amounts of milk, and large well-formed breasts from which milk spurts out in a jet are not infrequently disappointing in the smallness of their yield, which is found by daily weighing the amounts (Jäschke).

Milk shortage of the mammary gland is taken to be present when the child only drinks amounts which are very small or too small to measure, only a few drops flow out when pressure is applied to the gland, and only a few cubic centimetres can be drawn off with the pump after considerable difficulty. At first it is very difficult to determine whether it is only a case of a breast yielding with difficulty or whether psychical inhibitions are also concerned in the deficient secretion. Should the establishment of the secretion, which usually takes place on the third or fourth day, be delayed, then it cannot be a case of true hypogalactia depending on bad development of the glandular substance, it may be a type of delayed establishment of lactation. The secretion may not begin until the fifth or sixth day, at the end of the first or at the beginning of the second week. The delayed establishment may also be fairly sudden but usually in such cases the secretion comes on quite gradually by imperceptible stages and is not plentiful until the second or third week, there being no definite establishment of lactation. Schlossmann has brought forward the following example: a wet nurse yielded

| | | |
|------------------|---|---------|
| On the sixth day | — | 200 gm. |
| " 14th " | — | 380 " |
| " 15th " | — | 405 " |
| " 20th " | — | 595 " |
| " 30st " | — | 800 " |

The originally minimal amounts of fluid may increase quite gradually and although these women may not always supply enough milk to be able to feed their infants for some months on the breast alone, there are many who can furnish the basis for an "allaitement mixte" for one to three months; they can therefore hardly be reckoned among those incapable of suckling. Moreover it is often astonishing how well some glands function, which a few weeks before had to be considered as very poor in milk. In view of the occurrence of such cases it is hardly justifiable to give the mother a definite opinion as to her capability to suckle during her lying-in period (Hegar). The many statistics derived from maternity clinics concerning the capability to suckle therefore suffer from a defect which must not be underestimated; they tend to give too low figures, since many women who are incapable of suckling during the puerperium are able to do so later. The following examples illustrate this fact:—

(1) Delicate but healthy infant, weight 2,750 gm. At first marked hypogalactia. The child drinks barely more than 10-25 gm. per meal during the first week; neither can more be obtained with the pump. As much as could be obtained was pumped off and pressed out and given to the child with a spoon after the attempt at suckling. After feeding with small amounts of tea. Marked underfeeding; infrequent evacuation stools; weight at end of first week 310 gm. below weight at birth; a very considerable difference in proportion to the total weight. In the course of the second week the secretion improved, but the daily amounts still remained far under the average level (150-200 c.c.m.). The secretion then improved steadily, though slowly. The curve of weight rose continuously, but the weight at birth was not passed until the sixth week. After the sixth week one bottle of two-thirds milk, in the 24th milk a second, from the sixth month onwards porridge. Gradual weaning until the beginning of the 9th month. The child did not suffer any disorder whatever during its suckling period, and weighed 9,850 gm. at the end of the first year. This case demonstrates very clearly the lack of danger from underfeeding during the newborn period and shows that a breast originally decidedly poor in milk can develop into one capable of functioning adequately even for a long period of lactation.

(2) Elderly primipara with marked hypogalactia and retracted nipples, which at first could hardly be held. The underfeeding reached such a degree that from the seventh day and during the next week 100 gm. of human milk drawn from another woman was given daily, besides this, a considerable amount of tea was given. The mother's breast was systematically emptied with the pump, though only very small amounts were obtained. Beer's method of hyperæmia was also tried for some time. It was not until the third week that the child took the breast and was able to suck by means of a Soehnle's nipple shield. In the fourth week, on account of becoming progressively thinner, it was fed with half milk with nutritive sugar. In the fifth week the breast was so far improved that the child was able to drink without a nipple shield. From this time it was given three meals from the breast and three bottles of half milk. Under this allaitement mixte—the quantities taken each time from the breast amounting to 100-150 gm.—the child progressed excellently and appeared like a healthy breast-fed child. The weight, which at the end of the seventh week had only just passed the weight at birth, rose steadily, as was shown by weekly weighings. In the fifth month the feeds of pure milk were increased so that the natural feeding came to an end at the beginning of the sixth month. At the end of the sixth month the weight amounted to over 6 kg. This case shows that in consequence of continuous

attempts at suckling success may be achieved even under conditions which were originally most unfavourable; it is true that this involved a considerable degree of underfeeding, but did not have any deleterious effect on the further progress of the child.

The meaning of "capability to suckle" is very vague. We believe to-day that an exclusive diet of milk (even of human milk) throughout nine months is not only unnecessary but also not desirable, at least not in all cases; nowadays it is usual, even when the mother's milk is plentiful, to give other food as well after the sixth month. Even though the capability to suckle for nine months be considered as the normal capability for suckling (Bjelenky)—this is far too drastic an interpretation of the term which is of little use in practice. It would be equally incorrect to regard only such women capable of suckling whose children regain their weight at birth after 8 to 10 days.

If the nutritional needs of an infant exceed the supply it derives from the milk of its mother before the end of the first half year and if the additional feeding with cow's milk be found necessary even earlier (after about three months), such a mother must certainly not be classed as incapable of suckling. Infants whose nutritional demands have to be met during the first three months with one to two bottles of cow's milk mixture, besides breast feeding, should rather be counted among the breast-fed than among the artificially fed infants. From the practical point of view every woman can be described as capable of suckling who during the first year can feed her child chiefly on her own milk.

Only the different interpretations of the expression "capable of suckling" are able to explain the enormous differences shown by the statistics dealing with this subject; the following table from Sueda illustrates this point:—

| Twilagen | 1901 | 79 per cent. capable of suckling. | | | |
|------------|---------|-----------------------------------|---|---|---|
| Zürich | 1904 | 66-70 | " | " | " |
| Halle | 1900 | 25.3 | " | " | " |
| Stuttgart | 1901/02 | 100 | " | " | " |
| Strasbourg | 1904 | 42.4 | " | " | " |
| Halle | 1895 | 55.2 | " | " | " |
| Freiburg | 1891 | 33.3 | " | " | " |
| Freiburg | 1892 | 45.9 | " | " | " |

None the less it is necessary to distinguish various degrees of insufficiency, as Hegar does for instance, who distinguishes between fully capable, incapable, partially capable and totally incapable women. The capability to suckle must not be considered as exclusively due to insufficient development of the glandular substance, since in this connection various combinations with other difficulties of location may be of the greatest influence (e.g., weakness of sucking on the part of the child, bad nipples, illness of the mother, &c.).

It may be definitely asserted that no absolute incapacity for suckling rests on anatomical grounds; for all mammae, even the badly developed, contain secreting parenchyma, which in every case

may be utilized to provide food for the infant (Engel). But there is undoubtedly a diminished capability for suckling, which cannot be overcome in spite of all efforts and this diminution may be very considerable; so little mother's milk may be available that quantitatively it can only be considered as an addition to the main source of food.

It is not assurdly a very rare occurrence that an incapability of suckling renders necessary additional feeding with cow's milk during the newborn period, especially during the first weeks of life, in fact that period of infancy when feeding with human milk is of the greatest importance. Although Walcher's figures of 100 per cent. efficiency of lactation cannot be altogether relied upon it is none the less exceptional for the natural method of feeding to fail absolutely. Martin, supported by a large number of observations, is probably correct in stating that with very few exceptions a woman can suckle if she wants to or if she has to. He found that 99.3 per cent. of mothers could feed their infants with their own milk; also Jaschke obtained the high total of 97.8 per cent.; similarly, elsewhere, not much less favourable statistics were obtained if the figures be considered which concern children fed on mixed diet and women who could suckle at all, e.g., Halle, 97.6 per cent.; Munich, 95.2 per cent.; Strasburg, 94.6 per cent.; Würzburg, 92.7 per cent. (Dürig). Various other factors are also concerned on this matter. The capability to suckle does not appear to be the same in different districts and in different classes of society. It is at any rate very remarkable that Hegar should state that out of seventy-five women in his private practice six could not suckle at all and twenty-two only until the fourteenth day, and still more striking that when Meineri and Rietschel circulated the question to members of the German "Gesellschaft für Kinderheilkunde," thus among a class prejudiced in favour of suckling, in 41 per cent. of the women the attempts at suckling failed. These are facts not to be underestimated, but it is not improbable that if in the first attempts at suckling a better mastery of technique were shown than is possessed by many midwives and children's nurses and also by many women and children's doctors, better results would have been obtained.

A certain importance is without doubt attached to the age of the mother; for it cannot be denied that hypogalactia, delayed establishment of lactation and poorly yielding breasts occur relatively more frequently with elderly primiparae than with younger ones.

It has often been attempted to bring therapeutic measures to bear on the hypogalactia which is due to bad development of the glandular substance of the breast. It is difficult to decide on this point as in how far prophylactic measures have been successful. If, as is often supposed, the bad development of the breast depends on an inherited hypoplasia of the organ and on degenerative processes then a remedy is only to be hoped for when the improvement for future generations is begun by a widespread increase in

suckling. The way is not quite clear in which favourable influences may be brought to bear on the mammary secretion shortly before pregnancy. That good feeding and a hygienic mode of life are of primary importance, as one would assume *a priori*, is to a certain extent disproved by the better capability to suckle of those women delivered in maternity hospitals than in the upper classes of society, although the former are chiefly girls belonging to the hard working class and who often live under anything but hygienic conditions and do not always obtain the best food. Gruber says that the conditions of suckling in the upper classes would be improved if the young girls, like the cows, were driven out into the pastures; against this opinion it must be stated that young ladies who spend some months out of doors every year in the country receive far more of the advantages possessed by the cows (food, air and light) than do servant girls and working women, but experience teaches us that the latter are better able to nurse their own children. Possibly it is the regular bodily exercise which is of advantage for the development of the glands. Greater hopes may therefore be centred in the modern pursuit of sports for influences favourable to lactation.

The methods advised for the increase of the mammary secretion are very numerous. With regard to the galactagogues on the market which are more or less strongly advertised, it is the united opinion of the doctors who have observed their effects that their action is due more to suggestion in the mother than to stimulation of the gland. But this action must not be underestimated. When no result follows from the psychical treatment carried out by doctor and nurse, or if it be counteracted by the doubts and fears of anxious midwives, grandmothers and aunts, then a galactagogue covered with numerous testimonials of success plays the part of comforter and gives fresh hope to the previously despondent woman. It is of little importance whether preparations considered as specific galactagogues (Lactagol, Gallega, &c.) be given or whether nutritive preparations (Santalogen, Maltitropin, Maltyl, &c.) be substituted. When the latter improve the general state of nutrition they may also be good for the milk secretion, nevertheless it is very improbable that the improvement will take place as rapidly as is required, when there is a shortage of milk in the first days of lactation. The effects of a plentiful supply of fluid and good strengthening food during the puerperium are limited for this reason.

It is by no means impossible that in time some preparation, probably an organic extract, may be discovered to increase the secretion of milk. The experimental results previously given support such a hope; but at present no such substance has been discovered.

Sigmond calls attention to the researches of Hertoghe, who observed an increase in the volume of the breast and of the secretion on administration of thyroïdin. He gave thyroïdin during her second pregnancy to a woman who had been unable to suckle her first child (just three a day or so for several months), and successfully obtained the best results with it.

Since experience has shown that the emptying of the breast as far as possible stimulates the secretion, the child must be put to the breast regularly even when the amounts drunk are negligible. If the child has sufficient sucking power and inclination, the stimulation of sucking alone is certainly a galactagogue of considerable power. Should this not be the case it is possible that the application of another older infant may be useful, but it is necessary to add that such a child, if used to drink from a plentifully yielding breast is not always willing to suck at an empty one—therefore the result may be disappointing. Should no strongly sucking infant be available the secretion, however scanty, should be evacuated by frequent application of a milk pump or manual pressure. The recommendation to allow the husband to suck at the breast (Zloristi) may not accord with everyone's taste. Jacobius recommends that the mother should herself suck at the breast at regular intervals by means of a tube attached to an ordinary nipple shield.

In order to stimulate the circulation massage may be employed. According to Rummel this is particularly useful with strong turgid breasts; this is done by the breast being stroked and kneaded in a radiate direction, both before and after the application of the child.

A very excellent way of stimulating the secretion is Bier's method of hyperemia (Moll, Jaschke). A glass cup of 20 cm. diameter with wide everted edge is placed on the breast in such a way that it touches all round. The woman presses the cup, the edge of which has been previously well greased, on her breast. Suction is then made with an air pump or according to Moll with a water vacuum pump, which, apart from its cheapness, has the advantage of sucking the breast slowly into the cup as the vacuum gradually increases. Jaschke recommends that the first congestion should not last more than fifteen minutes, the succeeding ones twenty-five to thirty minutes. The degree of congestion necessary is difficult to decide; evacuation is continued until the milk squirts out from the breast in a fine stream. The number of cuppings per day should not exceed three.

Should the increase in the production be delayed in spite of every care, the question arises whether and how the natural food should be supplemented. In view of the low nutritive demands of the infant during the first week it should usually suffice to see that plenty of fluid is given so that too great drying up may be avoided. In consideration of the fact that with a small secretion the caloric value is high it seems quite rational to increase the concentrated diet by adding indifferent fluids. If human milk be available its addition is naturally the best to help over the worst days, but only such quantities must be given so as not to satisfy the appetite of the infant so completely that it gives up its attempts at sucking at the unproductive breast of its mother.

With regard to the availability of human milk the conditions in maternity hospitals are naturally very different from those in

a private house. In the former there is always sufficient human milk available to save the few infants, who cannot obtain enough milk from their mothers, from progressive under-feeding. The author is able to state quite positively from his own experience that in a maternity clinic it is hardly ever necessary to resort to feeding with cow's milk. An excess of milk which can be drawn off should always be at the disposal of women in maternity hospitals. In institutions on a smaller scale in which this may not always be the case, human milk preserved by the Mayerhofer-Prizibram method may be substituted for fresh milk. It is very desirable that feeding with pumped off human milk, which gives such excellent results with infants, should be much more widespread than is now the case. There is no danger of injury to the mothers by drawing off excess milk nor is it to be feared that the infants of the women in question would go short of food. The emptying of the breast, especially at the time of the establishment of lactation, even increases the comfort of the mother and is also of advantage to the child, since it is better able to suck at the softer breast. Moreover, the evacuation of the breast can but improve the secretion. The prevention of retention of milk is further a prophylactic against mastitis. The removal of milk is thus of advantage to the mother, to the child and to the children of women who have little milk. The popular custom in many institutions of the exchange of infants between women who have much milk and those who have little, is a procedure not to be generally recommended owing to the danger of carrying disease and at all events should only be permitted under careful supervision.

With regard to the method of removal of the milk the conclusion has been reached in institutions that with skill the manual method yields as much, if not more, than the ordinary breast pump. For the earliest period, however, this is certainly not the case since the breasts are usually far too sensitive to touch. At this time the milk pump is an absolutely indispensable instrument.

The varieties of breast pumps which have been put on the market from time to time are very numerous. They differ partly in the mechanism for sucking and partly in the shape of the receptacle for the milk. The sucking off is done either by the mouth of the mother herself or by means of an aspirator or with a suction pump. Auvard's aspiratory nipple shield, which is much in demand at the present day, as also its modification by Budin, consists of a spherical glass receptacle which is connected at its base by a broad opening with a funnel-shaped side tube; on both sides are small processes to which two rubber tubes are attached; through one of these the mother sucks her milk into the glass receptacle, the child drinks through the other (see fig. 23). The disadvantage of all such forms of apparatus is that saliva is very liable to get into the receptacle. This may certainly be prevented by the interpolation of a glass vessel, such as a Wulff's bottle, but the sucking in time becomes very tiring and the production is often very small.

The simplest form of breast pump is one with a receptacle provided with a glass cone with funnel-shaped side tube through which milk is sucked out by means of a rubber ball. Since the receptacle is difficult to empty and clean Ibrahim recommends a pump which may be emptied through an opening closed with a



FIG. 24.—Breast pump after Jaschke, with glass part after Scherbak.



FIG. 25.—Removal of milk with Jaschke's breast pump.

stopper. Modifications of Ibrahim's pump have been invented by Forest and Kaup. The glass part of Reyher's pump consists of a backwardly directed graduated tube, at the upper end of which at one side is the funnel-shaped side tube for the nipple, and on the other a smaller tube for the vacuum tube, on the lower end of the receptacle is a stopcock by which the vessel may be opened and the milk removed.

By far the best results are obtained by the use of Jaschke's breast

pump. The sucking takes place neither by the wearisome sucking with the mouth nor by an aspirator which involves continuous removal of the glass part from the breast and makes the pumping a very tiresome performance, but with a pump fitted with a valve which makes it possible to leave the glass part on the breast while moving the piston; in this way the thing is done in a speedy and comfortable way. Jaschke's pump will undoubtedly displace all the other types. Scherbak has produced a practical modification of the originally somewhat too large glass part of Jaschke's pump (figs. 24 and 25).

A cheap method of sucking off human milk can be done with the apparatus devised by Bock and S. Weiss. The procedure is as follows: a previously warmed bottle is filled with boiling water, this is poured out after a few minutes, the edge of the bottle is cooled by dipping it into cold water or by a cold damp cloth and placed on the breast; after eight to ten minutes the breast evacuates the milk by the cooling of the bottle. Bock uses an ordinary graduated bottle, Weiss has devised a "milk drawing bottle" with a small bell-shaped side tube.

The maternity institutions used by women of means could provide the human milk required by engaging a few good wet-nurses, who could be taken in together with their infants, milk being pumped out of their breasts as required. The mother's milk could be well paid for with far more justification than many galactagogues or milk preparations—thereby covering the cost for the wet-nurse's keep. It seems almost inconceivable that such an organization, which would make any form of artificial feeding superfluous during the first two weeks, has not yet been carried out. Why does not infant welfare, which is so ardent in the cause of breast feeding, do something for the newly born children, who need human milk more than all other infants but who are none the less fed superfluously with cow's milk day by day in wealthy circles on account of the difficulties of lactation?

Conditions are naturally most difficult in private houses. The institution of the so-called "wet-nurses" (*Stillfrauen*)—Brüning, von Stark—might well be helpful for these cases; with proper organization, at least in large towns, pumped off human milk in fresh or preserved condition (from wet-nurse institutions, maternity hospitals, &c.) should be available for use. The lack of such means, when the mother's secretion is insufficient after the first week and when a wet-nurse cannot easily be obtained, makes artificial feeding or the addition of cow's milk unavoidable.

With all the difficulties of lactation dealt with above and especially with hypogalactia, if the nutritional demands of the child cannot be approximately met with human milk and if one has not resorted to cow's milk a certain degree of under-feeding may result. It is obviously of great importance whether such a condition of under-feeding at the beginning of the period of lactation has any bad effects on the subsequent development of the child.

Under-feeding is first shown by an alteration in the curve of weight, the physiological minimum weight moves from the third or fourth day to one of the following days, the child thus continues to lose weight. Usually a condition is soon reached in which the weight remains constant, this lasts several days and then gives way to a slow increase; the sharp angular curve under normal conditions takes the form of a very gentle curve, or at a very low level of the curve irregular variations in weight are shown.

To what extent there is a causal connection between transitory fever and under-feeding in the first few days will be dealt with more carefully in the discussion on transitory fever (*vide infra*). Pies and Birk have shown that as a consequence of deficient nutrition during the first weeks of life symptoms of the exudative diathesis may arise. The children are strikingly tired and sleepy, their skin is dry and flaccid, very subject to injury, and has a tendency to furunculosis, intertrigo and eczema. With cessation of under-feeding these symptoms disappear. Langstein justly objects to the view that these symptoms of deficient nutrition of the skin are signs of a diathesis. At all events, it is not permissible to conclude from these experiences that under-feeding can provoke an exudative diathesis, a conclusion which would involve serious consequences with regard to the dieting of infants. That under-feeding can exercise a deleterious effect on the infantile organism, making it more susceptible to digestive disturbances and infections, is verified by clinical observations. Distinction must, however, be made between under-fed infants who are fed naturally or artificially, and according to whether the under-feeding goes so far that the curve of weight runs horizontally, or rises gradually, or whether a loss in weight takes place, and, finally, whether the child in question is a few days or two to three weeks old. In the first week of life, at the time of the most frequent difficulties of lactation, injurious consequences of under-feeding are hardly to be feared, owing to the small demand for food, and may be checked by an adequate supply of fluid. It is permissible, when an increase in the number of meals produces no increase in the amount drunk, to wait at all events until the second or third week, since, according to the experiences of the previously mentioned authorities, it is possible to disperse these symptoms by increasing the food, even after a still longer period of no increase in weight and after the appearance of skin symptoms.

The healthy infant possesses a very remarkable resistance against hunger, especially against the hunger for organic material, this representing an important criterion of health in consideration of the deleterious effects of hunger in wasted, atrophied and thus seriously affected infants (Rosenstern). When we consider that the healthy newly born child is an example of an undamaged organism, that under physiological conditions it has but a very small need for food, that hardly any inanition but only under-feeding is present, which with sufficient intake of fluid and the best quality of food

(human milk) does not even injure the sick infant, but even facilitates the regaining of health, we have quite sufficient reasons for estimating as trifling the dangers of under-feeding of breast-fed infants during the newborn period, and especially during the first week; it is especially at this time that the mother's anxiety that the child is in danger of starvation is the greatest.

Feeding with cow's milk during the first period of life is always a somewhat risky proceeding. Before the end of the first week it is not advisable because it always involves latent dangers, and because the temporarily good results shown by the satisfying of the child and of the mother often lead to the total abandonment of the natural way of feeding. The introduction of *allaitement mixte* is only advisable when the mammary secretion is to a certain extent in progress, and this may last one or two weeks. The addition of cow's milk may also be postponed until the second week, since during the first week even a marked degree of under-feeding may usually be borne without injury, and may easily be remedied. As a rule the additional feeding with cow's milk is not to be recommended until the mammary secretion is partially established, when there is a prospect of a successful *allaitement mixte*.

(C) ARTIFICIAL FEEDING AND *ALLAITEMENT MIXTE*

The total or partial feeding of the young infant with cow's milk is necessitated when no wet-nurse is forthcoming, and when the secretion of the mother's breast after waiting some time does not show any tendency to increase and remains so small that there is considerable danger of under-feeding the child; artificial feeding is also necessary when there is a contra-indication to suckling.

The dangers which the child would run in sucking from the breast of a sick mother consist of infections and intoxications. With regard to the former the questions concerning suckling will be treated in greater detail when infectious diseases are being dealt with. At this point we will content ourselves with the remark that the dangers threatening the child are due far less to the transference of pathogenic or toxic substances in the milk than to the infectious environment caused by the proximity of the sick mother. In this respect, most to be feared are those diseases transferred by the air and, in particular, tuberculosis. An open tuberculosis of the mother which may lead to infection of the air of the room is so dangerous for the child that suckling must be forbidden, since but a short contact with the mother may lead to fatal infection of the child. On this account open tuberculosis of the mother is the chief contra-indication for lactation. When it is impossible to remove the child from its parents' house, feeding is better done with pumped-off milk than with artificial nourishment. Cured tuberculosis, or such forms in which no free bacilli leave the mother's body, are no strict contra-indication to suckling.

Syphilis of the mother is no contra-indication to suckling when symptoms of the disease are not present in the child. Only in the rare cases in which a mother is not infected until near the end of pregnancy can syphilis be a reason for not feeding the child from the breast (Czerny-Keller).

Acute infectious diseases, especially acute exanthemata, are usually no contra-indication to suckling, because the child possesses a high degree of immunity to them. Erysipelas alone is much to be feared, in view of the susceptibility of the child for it; absolute and immediate separation from the mother is therefore advisable. In the course of severe and acute diseases the secretion of milk gradually diminishes. In some fever processes the secretion of milk is inhibited only at the beginning, often only during the first twelve or twenty hours (Mocher). In all infectious diseases of the respiratory tract (influenza, diphtheria, &c.) great care is certainly necessary, but the child may be protected from infection by suitable prophylactic measures. The milk in itself is not dangerous, and may therefore be given in the pumped-off condition if necessity arises.

Of especial importance is the question whether in septic puerperal conditions toxic substances can be transmitted through the milk, and in this way be injurious to the child. It appears that no great practical importance need be attached to this danger. If puerperal sepsis is so severe that an overflow of bacteria into the blood and milk is to be feared, the question of the advisability of suckling is usually settled by the condition of the mother being too serious to permit it or the amount of milk being too small. Transitory febrile diseases of the puerperium, which only moderately influence the general condition of nutrition of the mother, can be no contra-indication to suckling, even though persisting for some time.

On this question the author has a number of observations at his disposal. If one follows the temperature curve and general condition of infants in puerperal fever wards, it is practically never possible to detect signs of unfavourable influences on the child. According to the investigations of Basch and Weleminsky, bacteria only pass over into the milk when very severe lesions of the mammary tissues are present, such as seldom come under consideration; also the passage of toxic substances into the milk, although not disproved, is hardly probable according to clinical evidence. For this reason much less is to be feared from the milk than from the means of infection from the genital secretions of the mother, and prophylactic precautions are most necessary; premature forbidding of suckling may be much more injurious to the child than otherwise. In these cases also feeding with pumped-off mother's milk must be considered.

The possibility of a purely toxic injury of the newly born child by its mother's milk is chiefly furnished by eclampsia. In respect of this Goodall gives the following advice: If after delivery the

mother shows marked signs of toxemia or convulsions, then the child must not be suckled for some days, nor any pumped-off milk be given; only after the disappearance of the symptoms may the child be put to the breast which has previously been emptied by the pump. According to Frost, the milk of a woman suffering from eclampsia is richer in toxins than her blood, especially after an attack of convulsions. If on this account the milk is not safe for the child at the time of the symptoms of toxemia, it is quite certain that the danger of maternal eclampsia is essentially greater for the child *in utero*. If it is born healthy it will probably remain so, when fed by the mother after the toxic symptoms have disappeared. That the post partum persistence of albuminuria is a contra-indication to suckling is not generally maintained. The author has in such cases permitted suckling to continue undisturbed without there being any effect on the children. Should the toxic symptoms of the mother persist after delivery suckling is usually impossible on account of the severity of the condition of the mother. Usually the prohibition of suckling need not be much respected since, if the mother survives, the symptoms of eclampsia usually disappear in a few days.

Nervous diseases and psychical disturbances, nervousness, hysteria, epilepsy and psychotic conditions of the mother may in her interest or on other grounds make suckling impossible, but do not appear to have any influence on the quality of the milk.

Most paediatrists contend that there is an essential difference in the quality of the milk of different women. There are undoubtedly more statements than verified facts on this point. The bad development of some breast-fed children, and the large number of cases of dyspepsia and colic are almost always due to the children and not to the milk.¹

There seem, however, to be exceptions to this rule. Zappert and Jelles even report a case of the different action of the milk of the two breasts of the same woman; the milk of one side caused symptoms of dyspepsia which disappeared when the other side was used. A. Meyer was able to detect bile acids in the milk of a very jaundiced woman; the child vomited very frequently at this time.

Although, as we have seen, breast feeding may be dangerous for the child when the mother is ill, yet in the great majority of cases little is to be feared, but illness may, through its severity, be the occasion of giving up suckling in the interests of the mother, even if suckling does not exactly involve any direct danger to the latter. Whether suckling should take place in such cases depends on the amount of milk in the breast at the time, on the kind and degree of its yield, and especially on the presence of any difficulties

¹ NOTE IN CONNECTION OF PROOFS.—Langstein, Rott and Edlén (see p. 102), *loc. cit.* have shown that there are great individual differences in colostrum milk, the nutritive value varying in the different qualities.

of suckling, and last but not least, on the wishes of the mother and the pain she suffers while suckling the child. Should a woman be suffering from a disease of which the prognosis is bad or doubtful, then suckling will only be permitted in exceptional cases; should the prognosis be good in spite of severe illness, then, as a rule, suckling will be allowed, or at any rate the child may be fed with pumped-off milk.

Of greater practical importance is the question to what extent weakness, anemia and nervousness of the mother may be unfavourably influenced by suckling. Doctors are often too indulgent on this point. If one sees, as is frequently the case, how frequently delicate, pale women become even buxom during lactation, then one will *a priori* always advocate suckling. Zlocisti thinks that the woman will not be "too weak" if the child thrives; he believes that in cases where, in spite of apparently plentiful lactation the mother suffers, the child also will not thrive. Be this as it may, one must always be prepared to combat under all circumstances the prevention of suckling on account of the delicate constitution of the mother. The same applies to hysterical conditions and that usually described as nervousness. If a mother loves her child her nerves are likely to be much more severely strained by the incidents which follow in the train of artificial feeding in early infancy than if she suckles her child herself. This must be explained to the women, and they must be reassured that the unpleasantness often accompanying suckling during the first days will disappear within a short time. Marked psychoses and closely allied hysterical conditions may naturally be a cause for prevention of suckling or for its interruption after a short time.

It sometimes happens that women suffer from severe nervous excitement each time the child is applied to the breast; if they are not accessible to sensible psychical treatment, then it is usually better to discontinue suckling.

From the gynaecological side it has been shown that in persistent bleeding from the genitals immediate weaning may act therapeutically, also that sometimes tonic uterine contractions cease instantaneously when the child is weaned (Chrobak).

In cases like the last it has to be decided which is in greater danger, the child if it be prevented from suckling, or the mother if she has to continue. For example, a weakly or premature infant with an artificial diet would, in all probability, be seriously prejudiced, and the natural feeding should under no circumstances be given up on account of nervousness or weakness of the mother, and, if more important reasons be present, only in extreme cases; if, however, a healthier and to all appearances constitutionally strong infant is involved, then after a short time additional feeding with cow's milk may be resorted to, and if this meets with success artificial feeding alone may be continued. Some doctors are fanatical in their advocacy of suckling, and in their lack of sympathy for some mothers they may do more harm than good to their cause. One

should not always consider the extremes "to suckle or not to suckle," since intermediate courses must always be borne in mind. If we are unreservedly in favour of natural feeding of the newly born, it does not mean that every child must remain at the breast for six to nine months, but if it progresses well it may be given mixed or purely artificial diet at an earlier date, if this be desirable in the interests of the mother. Churchill advises the cessation of suckling if the mother loses weight, if her appearance alters for the worse, if sickness, loss of appetite, lethargy, tiredness or sleeplessness occur, and if neuralgia, lock or sacral pains, or dragging pains in the breast and shoulders arise.

A liberal point of view is also the most intelligent with regard to those women who do not like suckling and who are afraid of a limitation of their freedom. Most assuredly many women would more willingly make up their minds to suckle if it were said to them that if the child did well she would be partially relieved of her duties after a few weeks. According to Czerny, the difficulties of artificial feeding diminish day by day when the child is either wholly or partially on human milk. From the second or third month the best results follow in the majority of cases when a healthy child, hitherto naturally fed, is put on a diet of cow's milk. There is no point in keeping back this fact from the mothers.

From the above it will be seen that the absolute necessity of feeding a child exclusively on an artificial diet from the first day of life is but a rare occurrence. Besides the few cases in which the doctor is compelled, owing to illness or death of the mother, to feed a newly born child on cow's milk from the beginning, it nevertheless occurs in our own time (fortunately an age where suckling still has its joy) that a woman refuses from the beginning to suckle her child.

Our experience concerning the artificial feeding of newborn children is somewhat scanty. It applies in the majority of cases to infants who are not artificially fed until the second or third week; with regard to artificial feeding in the first week we know exceedingly little. It must unreservedly be admitted that it is possible to bring up a child from the beginning on cow's milk. Thus in his text-book Kassowitz describes the case of an infant which developed perfectly well in every respect on an exclusively artificial diet, and there is no doubt that such successful cases are by no means so very rare. The tolerance of different children towards the demands made on their system by the nourishment varies within wide limits. One often sees in the out-patient department excellent examples of infants, of which one bears to one's astonishment that they have not only never had human milk, but have been brought up in a way contradictory to all the rules of paediatrics. As these are probably cases of extremely tolerant individuals that develop wonderfully in spite of a purely artificial diet, it is difficult to form

an opinion how far the results of feeding depend on the methods used in the individual cases. With children of good constitution the most different methods appear to succeed. Good results are heard of with mixtures rich and poor in fat, rich and poor in carbohydrates, rich and poor in protein, rich and poor in salts, e.g., buttermilk, cream mixtures and "Barkhausmilch"; sugarless milk mixtures—malt soup; $\frac{1}{2}$ -milk, full milk, albumin milk, &c.

The ordinary rules dealing with artificial feeding of the newborn child are less the result of practical experience (clinical observations being so scanty) as of theoretical paediatric principles.

The most common procedure in artificial feeding is the administration of $\frac{1}{2}$ -milk, which is composed either of one part of milk and two parts of 8 per cent. milk sugar solution (Heubner's prescription) or a lesser amount of sugar is added. Langstein and L. F. Meyer recommend the following amounts and proportions:—

| | | | |
|------------------|-----|--|---|
| 1st day | ... | ... | ... |
| 2nd | ... | 50 c.cm. $\frac{1}{2}$ -milk | + 2 gr. sugar $\frac{1}{2}$ x 30 c.cm.) |
| 4th | ... | 120 " " | + 2 " " " 10 x 20 " " |
| 5th | ... | 150 " " | + 3 " " " 10 x 30 " " |
| 6th | ... | 240 " " | + 3 " " " 10 x 40 " " |
| 7th | ... | 300 " " | + 10 " " " 10 x 50 " " |
| 2nd week | ... | 350 " " | + 10 " " " 10 x 60 " " |
| 3rd | ... | 500—600 c.cm. $\frac{1}{2}$ -milk | + 20 " " " 15 x 100—120 c.cm.) |
| 3rd and 4th week | ... | 750 c.cm. $\frac{1}{2}$ -milk (with 20 " " | 15 x 150 c.cm.) |

grain)

The following observations of Cramer deal with the daily amounts drunk by an artificially fed newborn infant ($\frac{1}{2}$ -milk with addition of milk sugar):—

| Original weight | 1st | 2nd | 3rd | 4th | 5th | 6th | 7th | 8th | 9th day |
|-----------------|-----|-----|-----|-----|-----|-----|-----|-----|-----------|
| 1,400 gm. | 0 | 50 | 120 | 110 | 250 | 350 | 400 | 480 | 500 c.cm. |
| 1,120 " | 10 | 30 | 50 | 100 | 300 | 350 | 350 | 420 | 440 " |
| 1,350 " | 15 | 30 | 45 | 100 | 270 | 220 | 250 | 290 | 350 " |
| 1,240 " | 0 | 30 | 30 | 90 | 150 | 200 | 240 | 200 | 320 " |

Czerny and Keller also consider $\frac{1}{2}$ -milk with addition of milk sugar (one teaspoonful to 100 c.cm. of the mixture) as the most suit-

¹ Barkhausmilch (bakehouse milk) is merely diluted cow's milk to which cream and sugar are added and in which the protein is predigested by trypsin. That such predigestion is quite superfluous follows from the fact that even the embryo possesses proteolytic enzymes. The increase in fat is certainly better founded theoretically, as it rests on the wish to make the milk of the same quantitative composition as human milk (as in Riedel's cream mixture and in Götterer's fat milk, &c.). It has been proved however that milk mixtures relatively rich in fat are not in general superior to the ordinary diluted milk. In later infancy it may occasionally be advisable to increase the fat of the diet; this may easily be done by giving diluted cream or by adding cream to the food; in the newborn period however such an indication does not arise. Attention must be called to this fact, see "Barkhausmilch." It has a wide reputation as a means of nutrition for the newly born child. It cannot be denied that good results may be obtained with this preparation; but there is no doubt that it is no more effective than the usual diluted milk of the same caloric value. If much more expensive preparations enjoy a remarkable popularity among a large number of doctors, this is probably chiefly due to the fact that the prescription of a ready preparation causes no racking of brains.

able first diet for a healthy newborn child, and they consider the lower concentrations (1:3 or 1:4) recommended by some doctors for the first days of week (Biedert, Döbel) as unnecessary.

Since the usual $\frac{1}{2}$ -milk mixture even after the addition of sugar only contains 400 calories per litre (Finkelstein), the intake of nutriment always remains below the level taken by the naturally-fed child unless the amount drunk is very much increased in comparison to the latter. Since a too plentiful intake of fluid is not desirable and is usually rejected by newly born children at any rate in the first week, attempts have been made to use stronger concentrations from the beginning. Kassowitz found that a mixture of equal parts of milk and water, i.e., $\frac{1}{2}$ -milk, with further addition of sugar, was tolerated excellently even by a newly born child, provided that no more than five meals were given in twenty-four hours. Martan also gives $\frac{1}{2}$ -milk even in the first week of life; he prepares it by adding equal parts of milk and 10 per cent. sugar solution. He recommends the following scheme:—

| 1st day | 1 or 2 feeds | 5 gm. $\frac{1}{2}$ -milk |
|--------------|--------------|-----------------------------|
| 2nd " | 6 " | 8—12 " " |
| 3rd " | 7 " | 12—20 " " |
| 4th—5th day | 7 " | 20—40 " " |
| 7th—10th day | 7 " | 45—60 " $\frac{1}{2}$ -milk |

The author has often had the opportunity of seeing that with correspondingly careful dieting even stronger concentrations such as $\frac{2}{3}$ -milk do not harm a healthy infant, at least after the third week. French authorities (Budin, Variot and others) have even given undiluted cow's milk to newborn infants with good results. According to Heubner, the individual meals must be a little larger in these cases, but must be less frequent than with natural feeding.

| 1st day | 3 meals | 5—10 gm. full milk or only 1/2 |
|---------------------------|---------|--------------------------------|
| 2nd " | 4 " | 15—20 " " " |
| 3rd and 4th day | 5 " | 40 " " " |
| From 5th day on | 5 " | 50—60 " " " |
| Then gradually increasing | 5 " " | 70—80 " " " |

It thus appears, even apart from complicated feeding mixtures, that the most extreme methods are used.

The author feels bound to say that in general for the first days (about the first week) it is best for prophylactic reasons to give a fairly strongly diluted milk mixture such as $\frac{1}{2}$ -milk; the demand for nutrition is small, and the need for fluid is met just as well by a diluted mixture. During the first eight to ten days it is probably of little importance which grade of dilution is used, 1:1, 1:2 or 1:3; the degree of dilution is of less importance than the absolute amount of nourishment (Cramer). The curve of weight may proceed quite normally even on a diet of very low caloric value (see the observations of Cramer given on p. 102). Later, in the course of the second or third weeks, the change may unhesitatingly be made to $\frac{3}{4}$ - or even $\frac{2}{3}$ -milk. The increase of sugar should at first be a small one owing to the danger of irritation of the intestine

by fermentation; sugar may be entirely omitted during the earliest days, and during the second and third weeks not more than 3 to 5 per cent. should be added to the mixture. In this way the recently much discussed question is evaded as to which sugar is the most suitable to add to the food of an infant. There is no difference worth mentioning between the action of cane and milk sugar when they are given in small quantities, at least not to the advantage of the latter. Malt-sugar-dextrin preparations may be used even for the young infant and are of much greater value (Soshlet's Nahrungszucker, Loefflund's Nahrungsmaltose, &c.). Water is used first for dilution. There is nothing to be said against Doebeli's suggestion of adding rice-water or oatmeal-water to the milk after the second or third week of life. With regard to the number of meals the same rules apply as for natural feeding. Similarly with regard to the volume drunk, the example of the natural feeding should be followed without regard to the lower nutritive value of the diluted cow's milk.

Feeding should be carried on during the first two to three weeks in such a manner that the curve of weight rises daily from the physiological minimum weight even when the feeding is artificial; the steepness of the ascent is of secondary importance, but no further fall should take place. In most cases this object should be attained in the first week even on so low a caloric value as that furnished by 1-milk.

Unpleasant consequences of underfeeding, injurious effects of the milk diet, an incipient atrophy (loss of weight, emaciation, and the excretion of soap stools) do not appear until the end of the newborn period. Attempts must then be made either to obtain human milk feeding (wet-nurse) or to resort in the usual way to mixtures rich in carbohydrate, such as Keller's Malzsuppe (malt gruel) (Schellhe), buttermilk, &c., or to try albumin milk. We will not deal further at this point with the results and dangers of dietetics.

Feeding with the said carbohydrate mixtures may give good results during the first week or even from the first day (e.g., buttermilk + 5 per cent. cane sugar, according to Hoffa); also milk mixtures rich in fat, similar to the percentage composition of human milk may, in many cases, lead to good progress of the child, but for the sake of precaution this is not to be recommended. The results are certainly better at first than those obtained from feeding with diluted milk of low caloric values; but owing to the big demand on the tolerance of the delicate organism the danger of bad effects must be anticipated. The injurious effect is chiefly shown by intestinal irritation; it may be due to the acids which arise from bacterial fermentation of the carbohydrates or from the constituents of whey to which the young infant is peculiarly sensitive (Hellich). At all events, it is better at first not to try and obtain too quick an increase in weight. Finkelschein-Meyer's preparation of albumin milk, which is so useful in digestive disturbances of older infants, is also to be recommended for the feeding of healthy newborn

children. The success of albumin milk therapy depends on the prevention of intestinal irritation by the presence of casein, and also on the possibility of a relatively richer intake of carbohydrate without injury to the intestine. A newly born child may even be fed on albumin milk for prophylactic reasons. Benfrey has actually come to the conclusion that the feeding of newborn infants with albumin milk, with the necessary technique, is followed by better results than any other method of artificial feeding, though the children in question were one or two weeks old. The daily amounts of albumin milk should amount to 150 to 200 gm. per kg. body weight. The addition of sugar (preparation of maltose) should never be less than 3 per cent.; in general, one should begin with 3 per cent., and if there is no increase in weight and disturbances he abstains an increase to 6 to 8 per cent. should follow. It is possible by means of this method to avoid a condition of under-nutrition which may always be associated with certain dangers for the artificially fed child about the middle of the first month (L. F. Meyer).

Should no albumin milk be available, a similar and more easily prepared mixture may be used, such as Heim and John's casein-rich cow's milk, Freer's albumin-cream, milk, &c. At present the number of observations recorded is too small to justify the formation of a definite decision, but it appears as though the feeding on casein-rich cow's milk mixtures should prove particularly useful for any infants who have to be fed artificially from birth.

The milk mixtures which have been mentioned for exclusively artificial feeding are also advisable for *allaitement mixte*. As already emphasized, additional feeding with cow's milk is to be avoided, as a rule, in the first week; should it prove necessary or advisable it can be limited to one or two meals of 50 to 60 c.cm. of $\frac{1}{2}$ - or $\frac{1}{4}$ -milk. From the second week onwards the individual amounts may be increased to 100 to 120 c.cm. The number of meals with cow's milk is fixed entirely in accordance with the available breast milk, very often one to two bottles is sufficient for quite a long time. The additional feeding may also take the form of a drink of cow's milk after every meal from the breast; as a rule, however, it is better to keep the natural and artificial meals separate. The additional feeding with mixtures rich in carbohydrate (Liebig's and Keller's foods, Dutch milk), which often have very good results with somewhat older, under-nourished infants, is perhaps not without danger during the first three weeks, and is usually superfluous.

It must once again be emphasized that mixed feeding, even when the cow's milk mixture is in larger amount than the human milk, is followed by far better results than purely artificial feeding. *Allaitement mixte* is thus specially suitable for women who for social reasons are unable to feed their children during the day. Even the smallest amount of human milk is of priceless value to the young infant.

(D) WET-NURSE FEEDING

By far the best substitute to feeding at the mother's breast is wet-nurse feeding. The early introduction of artificial feeding is so undesirable that when the mother cannot or will not suckle, one should always urge that a wet-nurse should be engaged. Feeding by a wet nurse is also the best expedient when serious difficulties of suckling or any contra-indication to suckling are present, whether justifiable or not. We will not deal more closely with the choice or examination of the wet-nurse, and will only mention a few important points with regard to the newborn period. Wolf demands of the ideal wet-nurse the following qualities:—

- (1) That her breasts secrete a sufficiently large amount of milk of good quality,
- (2) That her health is perfectly sound,
- (3) That she has been delivered about three months before, and lastly
- (4) That her own infant is free from syphilis and has developed well.

With regard to the first point, relatively small demands will be made on a nurse chosen to suckle a newborn child, but one with only a small secretion will not be chosen, because the nurse in question will usually be needed for a considerable time. The third requirement, that a wet-nurse should not take on her duties until at least three months after delivery is made because of the danger of latent syphilis of the child; since "a child which shows no symptoms of syphilis by the end of the third month is in all probability free from syphilis, and usually permits of the conclusion that the mother is also free from syphilis or has at least passed the infectious stage." There is, however, some incongruity between the period of lactation and the age of the child. For the newly born child colostrum or early milk, in consideration of its chemical and biological peculiarities, is undoubtedly the only physiological nourishment in the strict sense of the word. None the less the results of feeding with human milk from a later period of lactation have been thoroughly satisfactory even for the newborn child. The claim, therefore, that the child for whom the wet-nurse is required should be approximately the same age as her own child has been waived. But two points must be borne in mind:—

- (1) That the newborn child may easily drink too much from the plentifully secreting breast. This may usually be avoided easily by careful limitation of the number and duration of the meals.
- (2) That on account of the small amounts of drink required by the newborn child the breasts of the wet-nurse may show signs of engorgement with its ensuing results. Undoubtedly the best way of avoiding these troublesome conditions is to allow the child of the wet nurse to suckle; as this for many reasons is not always possible, care must be taken that the excess milk must be squeezed or pumped off several times a day.

Clinical observations dealing with the progress of newborn children suckled by wet-nurses are few. Gundobin reports, among others, on the comparative researches of Fessenko, in which the children suckled by wet-nurses were in the same condition as those fed by their mothers, and mentions the following three observations from Saddofsky. The three women concerned had suckled their own infants for two to three weeks after confinement, and had then become wet-nurses for other infants.

| Case | Day | Child | Amount drunk in 24 hours | Increase or decrease in weight | Loss in weight in feces and urine |
|------|-----|-------|-----------------------------|--------------------------------------|---|
| I | 1 | Own | — | —158 | — 50 |
| | | Other | — 37 | — 60 | — 37 |
| | 2 | Own | — 42 | —124 | — 46 |
| | | Other | —104 | + 16 | — 53 |
| | 3 | Own | — 32 | — 32 | — 23 |
| | | Other | — 81 | + 49 | — 9 |
| II | 1 | Own | — 5 | — 79 | — 53 |
| | | Other | — 96 | — 50 | — 78 |
| | 2 | Own | — 4 | — 80 | — 30 |
| | | Other | —207 | — 37 | —127 |
| | 3 | Own | — 52 | — 33 | — 23 |
| | | Other | —204 | + 57 | — 24 |
| III | 1 | Own | — 9 | —174 | — 52 |
| | | Other | — 42 | —167 | —160 |
| | 2 | Own | — 49 | —136 | — 82 |
| | | Other | —130 | — 49 | — 84 |
| | 3 | Own | —106 | — 35 | — 50 |
| | | Other | —199 | — 32 | —128 |

From the above figures it appears that the children fed by wet-nurses were in better condition, but that they only took comparatively small amounts of milk on the first day in spite of a plentiful supply of milk. It is also of interest to note that in spite of the increased amounts of drink the children none the less showed a loss in weight.

The following case gives a good idea as to the amounts drunk by the newborn child at the breast of a wet-nurse:—

| | | |
|---------|-----|--------------------------------------|
| 2nd day | ... | 40, 20, 20, 30, 40, 50, 50 = 210 gm. |
| 3rd | " | 40, 20, 40, 40, 15, 40, 45 = 270 " |
| 4th | " | 40, 60, 60, 60, 10, — = 270 " |
| 5th | " | 35, 60, 40, 90, 40, — = 245 " |
| 6th | " | 35, 50, 50, 70, 60, 70, — = 345 " |
| 7th | " | 80, 60, 70, 80, 70, 70 = 470 " |
| 8th | " | 50, 60, 70, 50, 60, 70 = 395 " |

The amounts drunk are only comparatively large during the first two days of suckling. During the following days, in spite of the very considerable demand, they hardly exceed the amounts of mother's milk taken by the normal child. The child in question was thoroughly healthy and weighed 4,000 gm. at birth. A week later it weighed 270 gm. less; from then onwards its weight increased steadily and during the following months it developed splendidly in every way. The weight at birth was not passed before three weeks. The wet-nurse was in the fourth month of lactation.

PART III

The Premature Child

A CHILD born before the normal end of pregnancy is termed premature. On account of the premature end of pregnancy the child reaches extra-uterine life in a state of development which is, under normal conditions, passed through in utero. The backwardness of the functions which the full-term infant shows under physiological conditions, is considerably more marked in the premature infant according to its stage of development. It is this state which gives rise to the fact that in comparison with the full-term infant the premature infant always shows a certain degree of "defective vitality," and a lower power of meeting the demands of extra-uterine life. It is, however, only a case of "relative" defective vitality, which is due to immaturity of the organism alone and which must be clearly distinguished from the absolute defective vitality dependent on pathological causes.

In order to elucidate this conception, Pfäundler has contrasted the curve of the vitality potential of a healthy premature child with that of a full-term newborn. By the potential of vitality is meant, according to Escherich, the capacity appertaining to every being "to maintain its individuality by assimilation and energy exchanges, to grow and to reproduce." The increase of weight, which follows per unit of body-weight in the unit of time, may serve as expression for this potential of vitality. According to Pfäundler, this valuation applies, strictly speaking, only to a potential of growth. It can be shown that in spite of the less steep rise in the curve of weight in the premature infant, the coefficient of growth may be not only no lower but even higher than that of the normal full-term child; that the premature child of "deficient vitality" if it is not ill, shows an even higher potential of vitality than is shown by the normal full-term infant. This is quite intelligible if it be borne in mind that the physiological curve of the vitality potential falls uninterruptedly during both intra- and extra-uterine life; the (healthy) premature infant is younger, dating from conception, and is on this account better equipped with the potential of growth. If the curve of the potential of the premature infant be displaced in such a way that its age is calculated not from the end of labour but from the time of conception, then a curve will result which very nearly coincides with that of the normal child. From this it results that the defective vitality of the healthy

premature infant only represents a relative idea and is due merely to the premature occurrence of extra-uterine life. The earlier in its development the foetus, accustomed to an intra-uterine life, leaves the uterus, the greater is its relative defective vitality and the less its suitability for life, although the organs are fully able to exercise their appropriate functions. To the relative defective vitality of the premature child are added in many cases an absolute *debilitas vitæ* or a pathological disposition, since premature birth often results from illness of the mother or of the foetus, so that the latter may be born in a more or less damaged condition.

When a child is born at the normal end of pregnancy it may be described as "full-term;" such a child may usually be called "mature," but the two conceptions must not be regarded as identical (Frank). There are infants who are full-term but who are not mature, or cases in which the development does not at all correspond with the duration of pregnancy; for example, Ahlfeld describes a case in which a 1,590 gm. child was born in spite of a definitely proved duration of pregnancy of thirty-eight weeks. Even if the calculation of the duration of pregnancy only gives very doubtful and inexact information, it often happens that many infants who arrive at the date anticipated by their mothers resemble the premature child in size and appearance. Possibly many of these are cases, as Ahlfeld supposes, of inhibited development of the foetus on account of disease of the mother (e.g., chronic nephritis). Such infants also behave like premature ones in their further development, although by no means always like those suffering from absolute defective vitality; these then are children, who at the normal end of pregnancy and after normal duration of intra-uterine life, are born in an immature condition.

Although as appears from the above the condition "premature birth" (time idea), "defective vitality" (constitutional idea) and "immaturity" (developmental idea) are theoretically by no means identical, in practice in many cases they occur together and in the majority of cases can hardly be distinguished from one another by clinical symptoms, at least not without a long period of observation.

The causes leading to the interruption of pregnancy and consequent premature birth are manifold, and in individual cases can often not be even recognized with any certainty. One of the most important causes of premature birth is syphilis. Those children who are born with obvious symptoms of syphilis or reveal them within the first few days are almost without exception born prematurely, but also those who only show the symptoms after a latent period are often born before the normal end of pregnancy. Among other chronic diseases of the mother which cause premature birth, the following may be mentioned: tuberculosis, alcoholism, chronic nephritis and cardiac disease. Intoxications (phosphorus, arsenic, mercury and lead) have a similar effect. Also acute infectious diseases of the mother lead relatively often to premature birth.

especially scarlet fever, as well as croupous pneumonia, variola, malaria, measles, typhoid, &c. Premature birth may also be caused by local disease of the uterus, by disease of the decidua, endometritis or by anomalies in the position of the uterus. Finally, traumatic influences (falls, blows on the abdomen, &c., lifting heavy objects and physical over-exertion) are given as causes of premature birth. Twin pregnancy is a relatively frequent cause of premature birth. When artificial premature delivery is resorted to by the obstetrician the chief indication for this operation is afforded by contraction of the pelvis; far less frequently, other indications are involved such as diseases of the heart or lungs, nephritis, profuse hæmorrhage, &c.

The clinical symptoms of prematurity differ very considerably according to the month of foetal life at which birth has taken place. The nearer this approaches the normal end of pregnancy the less characteristic are the clinical symptoms, and the differential diagnosis between these and small full-term infants is then often impossible.

If it be realized that the weight at birth of full-term children varies between limits of 2½ to 4 kg. and more, and that the body-measurement is correspondingly variable, it will not be surprising to find that the body-measurement of premature infants also varies greatly. Oberwarth has collected from various authorities the following table of average figures for weight and length in the individual months of foetal life:—

| Age of foetus | Weight | Length |
|---------------|----------------|---------------|
| 4th month | 350—1,041 grm. | 28—37 cm. |
| 5th " | 565—1,408 " | 35.2—37.5 cm. |
| 6th " | 797—1,700 " | 37.3—40.5 cm. |
| 7th " | 1,265—1,994 " | 42.0—42.7 cm. |
| 8th " | 1,585—2,213 " | 39.6—47 cm. |
| 9th " | 2,424—3,700 " | 46.1—48 cm. |

Rummel gives the following average figures (after Allfeld and Hecker):—

| Age | Weight | Length |
|-----------|----------------|---------------|
| 27th week | 350—1,041 grm. | 28—37 cm. |
| 30th " | 565—1,408 " | 35.2—37.5 cm. |
| 31st " | 797—1,700 " | 37.3—40.5 cm. |
| 33rd " | 1,265—1,994 " | 42.0—42.7 cm. |
| 35th " | 1,585—2,213 " | 39.6—47 cm. |
| 37th " | 2,424—3,700 " | 46.1—48 cm. |

Neither weight nor length give definite information for the diagnosis of the age of a foetus. The figures usually given as intermediate between premature and full-term are 2,500 gm. and 47 cm.; but there are smaller full-term and larger premature infants. In general, the length is of more significance than the weight. According to Holzbach the majority of premature infants are less than 48½ cm. in length; children 50 cm. in length are practically always mature. Doubtful cases are between 2,000 and 2,500 gm. in weight and 45 to 48 cm. in body-length; lower values almost always indicate premature, that is, immature births.

It is similarly impossible to give reliable general figures for the absolute body-measurement. Of greater importance is the proportion between different parts of the body, especially between the circumference of the head and that of the shoulders; in immature children the shoulder measurement never exceeds that of the head (Frank). Since the lower extremities exhibit more rapid growth than the upper half of the body during the latter half of pregnancy, premature infants in the early fetal months have proportionately shorter lower extremities than mature infants.

If the body-weight be compared with the length of the body the resulting quotient ($\frac{\text{Weight}}{\text{Length}}$) amounts to 60 to 80 in the mature newborn child; in the premature child it is 30 to 50, and in those under 1,000 gm. it may be as low as 25 (Oberwarth). The cause of this low weight in proportion to length may be regarded as the thinness of the premature infant.

The small amount of fat in the subcutaneous tissues is usually striking on external examination of the body. The marked turgidity of the skin, so characteristic of the mature infant, is absent in the premature. The skin usually lies over the bones and muscles as a thin covering without much panniculus adiposus, sometimes it is wrinkled and sometimes stretched and shining. The latter occurs when either sclerodermic or sclero-oedematous changes are present, such as is almost always the case with small premature infants with very low temperatures. The colour of the skin varies considerably. Usually icterus and erythema are particularly marked in premature children and they usually persist longer than with the mature child. If depression of the temperature occurs the hyperemia also usually disappears. A cyanotic coloration of the skin may easily occur owing to the frequent disturbances of respiration. The lanugo hairs are usually very plentiful and are particularly obvious not only around the shoulders but also on other parts of the body, especially on the face, forehead and cheeks. Miliaria may often be found on the nose of the premature infant. The auricle is sometimes flabby on account of the thinness of the cartilage and lies close to the skull. The nails of the fingers and toes are in many cases imperfectly developed and do not reach to the tip of the digit; this is, however, by no means a constant symptom, many small premature infants have well-grown nails which may even extend beyond the tips of the digits. The mammary glands of the premature infant are usually very minute or not palpable at all; as a rule no secretion of milk occurs. In immature girls the labia majora cover the labia minora, in immature boys the testes sometimes lie somewhat higher than the average for the mature child; since the testes may have descended as early as the seventh month this symptom is very often absent. Defects in the skeleton are usually not to be found in the premature child; softness of the skull is perhaps even more rare than in the mature infant. The size of the fontanelles also is equally variable. A certain backwardness is most noticeable in the thorax, which may be shown by its peculiar

softness and lack of resistance during respiration. According to Holdbach X-ray observations with regard to the condition of the ossification centres may give valuable information for the diagnosis of the maturity of a child. De Vearis calls attention to the peculiarities of the blood. The above signs of immaturity occur in varying number in different premature infants and definite conclusions in doubtful cases can hardly be drawn from their presence or absence.

The backwardness of the vital functions, which is shown more or less obviously in all the various systems of the premature child, finds its chief clinical expression in the condition of the body temperature. The thermolability, characteristic of all newborn children, is particularly great in the premature infant and lasts much longer. It is particularly the tendency to low temperature which dominates the clinical aspect of the case. In the fall of temperature on the first day of life (the temperature at the time of birth is at least half a degree lower than that of the full-term infant) an extraordinarily low figure is often reached; temperatures of 30° to 31° are by no means rare and sometimes the temperature falls still lower—the children may actually feel as cold as frogs. The extreme hypothermia of the first day can by no means be considered as a sign of defective vitality, this conclusion only being justified if low temperatures of 32° to 34° persist in the following days in spite of the application of heat. In the viable premature child it is possible by suitable application of warmth, to raise the temperature during the first days of life at least to 35° to 36° and to maintain it subsequently at a higher level. Should the application of warmth be interrupted the tendency to hypothermia expresses itself again in a fresh rapid fall in temperature. If the warming be carried out carelessly hyperthermia may also occur and the temperature of the child rise to 38 to 39° , but such overheating usually only occurs in somewhat older or relatively well developed premature children. During the first days high temperature of the environment often has as little effect as any internal agency which otherwise produces fever; infectious diseases are particularly often unaccompanied by fever in the premature child.

The thermolability is a result of the insufficient heat-regulating mechanism of the skin. The surface of the body of the premature child is larger in proportion to its mass and therefore the opportunity for heat loss relatively greater and is also considerably furthered by the defective formation of the subcutaneous fatty layer. The particularly small intake of nourishment in the first days may also be of some importance, although it is hardly probable that a relatively larger intake of combustible material, after a fixed minimum has been attained, will have any considerable influence on the body temperature; the human body is not a stove which may be heated much or little according to taste; as a rule the oxidation processes cannot be increased beyond a certain degree by the intake of substances which can be oxidized. By sufficient

intake of food the organism can only be prevented from attempting to counteract the heat deficiency by burning constituent parts of the body. Babak was able to show that the utilization of oxygen increases the more the temperature of the body falls, which shows that to a certain extent a chemical regulation takes the place of a defective physical one. (It is probable that toxic products may arise by these oxidative processes.)

Although we find the most important cause of hypothermia in the increased heat loss which arises from the insufficiency of the physical heat regulation, we must also realize that the thermolability of the premature child is essentially due to the functional backwardness of the central nervous system governing the complicated processes of heat regulation.

The functional backwardness of the central nervous system is shown particularly by the striking quietness of the premature child. Many premature infants lie in a condition resembling sleep which is rarely, if ever, broken by spontaneous crying; especially in the first days of life this quietness is an invariable phenomenon in small premature children. Outward stimulation, however, produces a reaction in the viable premature child. They cry, although often with a very weak voice which often resembles the "peeping" of a young bird; they also make movements of protest. In general all movements have a peculiar inert character, they take place slowly in worm-like manner, not so quickly as the movements of full-time children. If the outward stimulation ceases, then the child falls again into its somnolent condition.

The most important symptom resulting from the defective development of the central nervous system is the imperfect functioning of the respiratory centre. While in general the heart of the premature infant functions well and primarily, as a rule, does not involve any danger to life, disturbances of respiration are perhaps the most frequent cause of death or at least of dangerous symptoms. The breathing of small premature infants usually takes place quite superficially and very irregularly. The result is that the imperfect expansion of the fetal lungs is only overcome very slowly and imperfectly. The gaseous exchanges are very insufficient and excess of carbonic acid in the blood results, which for its part is not able to stimulate the inexcitable respiratory centre. Finally the cyanosed child entirely ceases to breathe, only after an astonishingly long interval (a minute or more) the threshold of stimulation is passed and breathing again takes place; the condition thus resembles the mechanism of Cheyne-Stokes breathing.

Such attacks of asphyxia and cyanosis, which sometimes take place without rhyme or reason, are particularly liable to occur in premature infants during the first week. If they occur in spite of suitable therapeutic measures and at progressively shorter intervals they constitute a very ominous symptom, and in one of the attacks death finally takes place. If they only occur occasionally, or become less frequent, then they may be overcome. The cyanotic

attacks are very often started by purely mechanical means, by the aspiration of food or vomited matter. The ease with which aspiration occurs is another result of insufficiency of the nervous system, namely of defective reflex function. If such a child swallows the wrong way while drinking or if it vomits, the fluid passing into the entrance to the larynx produces no cough reflex but flows down the trachea undisturbed.

Budin associates the attacks of cyanosis with underfeeding and quotes examples in which the attacks disappeared as the nutrition increased. It is quite conceivable that an improvement in the general nutrition would also improve the functions of the respiratory centre, but practical conclusions can hardly be drawn from such experiences during the first few days of life, owing to the difficulties in feeding, which are often very considerable. The attacks of cyanosis are most likely to occur after feeding, either directly after or somewhat later. If there is no mechanical hindrance to respiration the disturbance in respiration may be due to the inhibition of respiration caused by swallowing, thereby diminishing the otherwise small gaseous exchanges, so that a deficiency of oxygen results; the filling of the stomach may also possibly interfere with the diaphragmatic respiration (Birk).

Pulmonary atelectasis is dangerous to the child on other grounds. It is frequently the seat of infection, the pathogenic organism either entering the lungs by inspiration or by aspiration. Pneumonia in an imperfectly dilated lung is a very frequent cause of death in the premature child, without there being any reason on this account for assuming any particular weakness of resistance in general.

The function of the heart in the attacks of asphyxia is usually only secondarily involved. Should the attacks of cyanosis become more frequent the heart sounds finally become strikingly slow and dull though the heart may beat for some time after the respiration has finally ceased. The condition in the lungs due to this atelectasis may delay the closure of the foetal communications, especially of the ductus arteriosus (Rommel). The cyanosis may thus be increased indirectly by the heart.

A fragility of the blood vessels is often noticeable without there being a specific constitutional disease (syphilis) obviously owing to the delicacy of the young tissues; this is shown particularly by the fact that in the premature child traumatic, especially intracranial haemorrhages, readily occur.

Our knowledge is comparatively small with regard to the functions of the alimentary canal of the premature child. The presence of a defective function of the digestive enzymes as has been supposed by many authorities and made the reason for feeding with all kinds of predigested food mixtures, is most improbable, owing to the fact that most of the ferments necessary for digestion are already present in the foetus. None the less it may be assumed with all probability that the intestinal wall, which is imperfect at first even in the full-

term child, will be still more defective in function in the first period of the life of the premature child. The metabolic functions in general seem likewise not yet to be present in sufficient degree; the difficulty of rearing premature infants on artificial diet seems to point to such a conclusion.

Czerny and Keller mention researches of Chatriu, Noblecourt and Lemaire concerning the amount, specific gravity, chloride and phosphate content, cryoscopic constants and tonicity of the urine of premature infants; they add, however, that these investigations are of no value for the understanding of the processes of metabolism.

The nutritive requirements of premature infants are generally relatively high. While for the normal infant in the first period of life an intake of food of, at most, 100 cal. per kg. body-weight is considered sufficient, the energy quotient of the premature child is essentially higher, 110 to 120 cal. (Czerny-Keller); 120 to 130 cal. (Oppenheimer, Langstein-Meyer); 130 to 150 cal. (Salge); 115 to 150 cal. (Samelson). Similarly the premature infants observed by Birk and Meyer fed with human milk or suckled at the breast, drank amounts corresponding to an energy quotient of about 140 to 160 cal.; though the above authorities do not think that these amounts justify any conclusion concerning either the optimum amount of nourishment or the minimal needs, and that amounts of 100 to 110 cal. are as a rule adequate to guarantee also the physiological development of a premature child. Correspondingly a correction must be made concerning the volumetric measurement of the food requirements; according to Bodin premature infants up to 2,500 gm. drink after the tenth day of life on an average one-fifth of their body-weight (or 200 gm. per kg.), while mature infants in the first three months require about one-seventh of their body-weight (or 140 gm. of human milk per kg.). The observations of Birk and Oberwarth indicate that premature children also, especially the heavier ones, are able to thrive on a supply of nourishment which only amounts to a sixth or a seventh of the body-weight.

It is for many reasons easy to understand that premature infants in general have somewhat greater nutritive demands than mature ones, or that at least these correspond to the upper limit of the demands of the mature infant. Since the energy requirement of an organism is proportional to its surface the premature child requires a larger supply of energy in view of the large size of the surface as compared with the weight (Heubner). Since moreover the caloric requirement of the infant diminishes gradually with increasing age, it is quite comprehensible that the premature child, who represents an earlier stage of development, will require a relatively higher energy quotient; the previously mentioned calculation of Pfandler, that the healthy premature infant has a higher potential of vitality is fully in accordance with this consideration. It must also be taken into consideration that a direct parallel cannot be drawn between the body of a premature child and that of a mature one, on account of the deficiency of the subcutaneous fatty layer in the latter: 1 kg. of a plump mature infant is not equivalent

to 1 kg. of a thin one; a fact which is often neglected in the calculation of the nutritive requirements of older infants; thus there are beyond doubt children who thrive well during the first months of life on an energy quotient of less than 100 and on the other hand there are thin atrophied ones who do not begin to increase in weight until the energy quotient exceeds 100. The total weight of a premature child includes a relatively larger amount of growing tissues than that of a mature infant. It must finally not be forgotten that in the artificial feeding of premature children the kind of food may be of importance in calculating the caloric value, this being on account of the possibility of fat not being utilized so well by the premature child as by the mature one (Birk).

What has been said about the nutritive requirements of premature infants is only applicable from a certain age onwards and does not apply to the very first period of life. Just as in the case of the mature child, special rules apply to the food required by a premature child during the newborn period. The energy quotient of 100 cal. is hardly ever reached before the second or third week and still less exceeded. Even under favourable circumstances the amounts of milk drunk in the first week only correspond to an energy quotient of 30 to 50. The body-weight relatively often shows a fall lasting over the third or fourth day or a period of equilibrium lasting several days, or small variations at the same level; in other cases in premature children, just as in mature ones, an increase in the body-weight takes place in spite of the very small intake of nutriment, which in this case also can only be explained by the retention of water. The small amounts drunk during the first week, the slow rise to the actual demands until the second or third week, must undoubtedly be explained as physiological; on this point the premature infant fully resembles the mature. Should it be a case of a child who is strong enough to suck at the breast or one whose food has to be supplemented, or is entirely artificially fed, then increase in the number of meals but rarely succeeds during the first few days of feeding; on increasing the amounts of food above a relatively low level the child either refuses from the first to take more or it vomits.

The following table from Oberwarth shows the amounts of milk for the first ten days which were drunk by premature children of different weights and calculated according to averages:—

| Day | Under 1,200 gm. | | | 1,200—1,600 gm. | | | 1,600—2,000 gm. | | |
|------|-----------------|--------|------|-----------------|-------|--------|-----------------|--------|--|
| | Bodin | Perret | Birk | Oberwarth | Bodin | Perret | Bodin | Perret | |
| | gm. | gm. | gm. | gm. | gm. | gm. | gm. | gm. | |
| 1st | 115 | 52 | 50 | 59 | 125 | 120 | 180 | 155 | |
| 2nd | 160 | 122 | 96 | 108 | 175 | 175 | 240 | 205 | |
| 3rd | 210 | 152 | 124 | 160 | 225 | 247 | 305 | 290 | |
| 4th | 225 | 200 | 151 | 120 | 268 | 281 | 355 | 345 | |
| 5th | 250 | 224 | 177 | 145 | 324 | 312 | 370 | 365 | |
| 6th | 280 | 230 | 191 | 105 | 335 | 347 | 375 | 390 | |
| 7th | 285 | 265 | 230 | 160 | 350 | 364 | 385 | 400 | |
| 8th | 310 | 281 | 243 | 240 | 380 | 391 | 415 | 435 | |
| 10th | 320 | 305 | 240 | 228 | 410 | 405 | 425 | 438 | |

Delestre with a somewhat different grouping obtained similar figures by weighing 140 breast-fed infants:—

| Day | Under 1,500 gm. | 1,500—4,500 gm. |
|------|-----------------|-----------------|
| 2nd | 125 gm. | 100 gm. |
| 3rd | 235 " | 230 " |
| 4th | 250 " | 260 " |
| 5th | 255 " | 350 " |
| 6th | 360 " | 360 " |
| 7th | 425 " | 325 " |
| 8th | 245 " | 330 " |
| 9th | 435 " | 340 " |
| 10th | 250 " | 345 " |

With small premature infants of 1,000 to 1,200 gm. or less, it is necessary to be contented during the first days of feeding with a 24-hourly amount of 30 to 50 gm., only in the second half of the first week are higher figures reached. Cramer recommends that during the first 10 days the size of the individual meals should be between 10 and 35 gm. and should never exceed 40 to 50 gm. With small premature children it is often necessary to begin with meals of 5 gm. It is impossible to make hard-and-fast applicable rules on this point, owing to the great differences in development of various children.

In many cases of premature birth the technique of feeding presents considerable difficulty. If it be a case of a relatively strong child weighing about 2,500 gm. at birth then the same rules apply as to the mature child. If the sucking power of the child be sufficient and if there be no disproportion between this and the ease of the yield of the mother's breast, then the child should be applied to the breast five to six times a day, and no differences in the mode of feeding will be made between this and the full-term child. In the small premature child also it does not seem necessary to begin feeding on the very first day. If it be considered advisable very small amounts of fluid may be administered. If the child be relatively weak at sucking and drinks so little at each meal that the total quantity remains considerably below the average level, then the number of meals may be increased to seven or eight, or, if the child does not refuse a larger quantity (and this is fairly frequently the case) feeding with pumped-off milk in a bottle may be resorted to. This however is not applicable to the first and second days of suckling on which the child can often not be induced to suck more than four or five times a day. All difficulties of lactation due to the peculiarities of the mother's breast (difficulty of yield, rigidity at time of establishment of lactation, bad quality of the nipples) are of increased importance in the feeding of premature children; it is much more often necessary to bring the previously mentioned remedies to bear upon the defects. With a breast relatively rich in milk and nipples difficult to hold, suckling with a nipple shield also frequently succeeds better than the direct method with the premature infant. Administration of pumped-off milk from a bottle often proves necessary with the premature child during the first days

of feeding; since the maximal suction which the child can bring to bear often does not reach the negative pressure necessary for the emptying of the breast (Cramer).

If the child be able to suck strongly at the bottle then, provided that sufficient pumped-off human milk is available, the difficulties of feeding are comparatively easy to overcome. They only become serious when the sucking power of the child is also insufficient for the bottle. Many premature infants tire after a little sucking when they have swallowed about 5 gm., and cannot be induced to suck again. In such cases the number of the meals must be increased. There is no reason for producing in such children a progressive exsiccation and inanition by maintaining the infrequency of the meals, when with eight to ten or even more meals a day a partially sufficient quantity is reached. A similar procedure is necessary when the child vomits larger amounts of food. The vomiting and regurgitation of the milk drunk, which is so very dangerous on account of the risk of aspiration, may often be prevented by hourly or two-hourly feeds of only 5, 10 or 15 gm.

If a premature child does not suck at all, either because it is too weak or the sucking reflex cannot be elicited, in such cases the



FIG. 25.—Medicine spoon, after Kermauner.

milk is poured into the mouth by means of a spoon, a pipette or boat-shaped vessel; the spoon devised by Kermauner, with its front end beak-shaped, is very practical. Often swallowing movements are produced if, instead of being put in the mouth, the milk is carefully poured up the nose. In these cases also it is necessary to begin with very small amounts.

When the child not only does not suck but also does not swallow effectively (probably from defective excitability of the swallowing centre in the medulla oblongata), when the milk poured in flows back out of the mouth, when the pharyngeal reflex is also absent and the milk which has been poured in threatens to flow down the trachea, then it is necessary to resort to feeding with a stomach tube. Certain authorities have been averse from this method, recommended by French authorities, because there is danger of overfilling of the stomach, disturbance of respiration and injury to the mucous membranes. Rott, however, was able to obtain very good results by this method, and the author has also had similarly satisfactory experiences. An ordinary Nelaton's catheter is used (about No. 14) about 25-40 cm. long, in which the distance from the epigastrium to the glabella is marked by a small strip of sticking

plaster. The tube which is furnished with a funnel is filled with milk (pinched by a second person or with a pinch-cock) and is introduced into the stomach up to the mark. The child is placed on its back, preferably with the head a little lower. The introduction usually takes place very easily without any opposition. The requisite amount of milk is then allowed to flow in, the tube is then pinched again to prevent drops falling, and the tube pulled out quickly. The amounts poured in vary according to the age of the child, and chiefly according to the limits fixed by the vomiting or regurgitation of the fluid after this mode of feeding; they amount at first to 10, 15 or 20 c.c., and may later be increased to 30 to 50 c.c.; the daily number of stomach-tube feedings amounts to five to seven, and under certain circumstances to eight to ten. With infants who are able to suck and swallow, but only drink very small amounts at each meal, a larger amount is given once or twice a day by means of a stomach tube. Attempts should always be made to change from one mode of feeding to the one next above it, thus from the stomach tube to the spoon, from this to the bottle, and from the bottle to the breast. Sometimes one sees after tube feeding for one or two days that a child is already beginning to swallow or to suck; bottle-fed infants usually soon begin to suck from the breast if they are persistently applied. The task of the nurse brings its own reward when it is possible to improve successively the method of feeding; it also falls to her lot to watch the child closely after drinking in order to treat promptly any attacks of cyanosis which are particularly liable to occur at this time. If every form of food be vomited human milk may be given as an enema.

To the difficulties of nourishment on the part of the child which are due to its weakness in sucking, to its defective requirements of drink, to the impossibility of giving it larger amounts of food, &c., must be added as a further difficulty the injury to the process of secretion in the mother's breast, which may occur on account of the insufficient emptying of the gland. It is a certain fact that the secretion of the mammary gland diminishes after a time, and finally ceases if not utilized; it must also be admitted that this danger is still greater when the emptying of the gland does not take place at the time of commencing lactation. In spite of this, it must be emphasized that the practical importance of this functional peculiarity of the gland is frequently very much overestimated; for a complete inactivity of such duration, as to stop the secretion of milk entirely, can always be prevented by appropriate treatment. Also without foundation is the fear that, on account of the insufficient emptying in the first days of lactation, the production of milk may be so greatly diminished as to cease when the child begins to suck after a few days; every gland which is only partially functional may easily be made to secrete, even though it has been quite insufficiently emptied for a week or more. There is, however, no need to let matters proceed to this extent, since we have means of causing the

establishment and its maintenance at a satisfactory level without involving the stimulation of the sucking child—namely, the mechanical evacuation of the breast with the pump or with the hand.

The following two cases illustrate this point:—

A 15-year-old primipara fed her premature infant in the first days with pumped-off milk, later with milk pressed out manually, for 21 months. The secretion was not only established and maintained in this way but increased to such an extent that the milk obtained was ample for feeding the child and there was always excess present. It was not until the tenth week of life that the child drank direct from the breast; from the third to the sixth month feeding was carried out exclusively in this manner.

The second case occurred in a 26-year-old primipara with a prominent congenitally cleft palate. In this case also the maintenance of the secretion was achieved first by pumping, and later by manual "milking" performed by the woman herself; so successful was this that the child thrived in a fully satisfactory manner on a diet of its mother's milk alone.

Helbich has reported quite analogous cases. He also was able to maintain fully the secretion of wet-nurses without the physiological stimulation of the sucking child, solely by artificial emptying with the milk pump. He was even able, with a badly secreting breast (also at the commencement of lactation), to increase the production by mechanical evacuation. The practical importance of such experiences is obvious.

A careful treatment of the secreting breast is also necessary when the child sucks directly. The small amounts of drink taken by a premature child are greatly out of proportion in any case at first to the power of production of the gland; the excess must be removed by pressure or pumping. If a healthy, strong child be available it may naturally be put to the breast, but this is not necessary. When a wet-nurse is engaged for a premature child it is naturally most suitable for the maintenance of the secretion to permit the suckling of her own child. Should this not be possible a diminution of the wet-nurse's milk may assuredly be prevented by artificial withdrawal of the excess milk.

Great difficulties in the feeding of premature infants arise in cases of delayed establishment of lactation or true hypogalactia. They are essentially the same as with the mature child, but with the premature child there may be much anxiety owing to the increased dangers of underfeeding. Thus, for example, Cramer believes (in contrast with similar occurrences in mature children) that it is dangerous with a premature infant to wait any length of time for the mother's secretion. For this reason he gives, if necessary, even on the first days, small amounts of cow's milk, and changes as soon as possible to human milk. During the first two to three days it is sufficient, even with premature children, to be contented with the administration of fluids. It must be mentioned at this point that with all difficulties in feeding injections of small amounts of normal saline or Ringer's solution (20 to 50 c.cm.) often have a decidedly beneficial effect, and should certainly not be

neglected in cases of milk shortage. If on the third or fourth day only very insufficient amounts of mother's milk be available, and if feeding with wet-nurse milk be impossible, and pumped-off human milk unobtainable, the introduction of artificial feeding or of allanement mixte cannot be avoided.

In spite of a few favourable results which were obtained with the artificial feeding of premature infants, the following statement of Czerny-Keller holds good: "It is the duty of every doctor who has to deal with a debilitated infant to use all his energy to obtain for it human milk during the first weeks at least. The dangers of artificial feeding are particularly great in the premature child; it must be described as a professional blunder when a doctor, without the most dire necessity, subjects a child entrusted to his care to these dangers."

If circumstances are such that the introduction of artificial feeding is unavoidable the procedure must, according to Oberwarth, be careful and circumspect. The choice of a suitable food mixture is not easy. As for the mature newborn child, the preparations recommended for the premature infant are most heterogeneous: ordinary diluted milk with addition of sugar, $\frac{1}{2}$ -milk (Oppenheim)—Oberwarth was able to rear a premature child weighing 750 gm. at birth with $\frac{1}{2}$ -milk—mixtures rich in fat and poor in casein (Pfaundler), also "Ramogen" mixtures (with slight addition of cow's milk) of 1 to 2 per cent. fat content (Neumann and Oberwarth); predigested milk preparations, milk peptonized, according to Badin and Michel, with fresh calves' pancreas, "Backhausmilch," &c. Czerny points out that according to the literature the artificial feeding of debilitated infants with peptonized milk appears to give the best results; he adds, however, that the information is lacking as to how many children have come to grief with this treatment, since nobody records bad results.

Of late years buttermilk has been very popular as a food for premature infants (Finkelschein, Birk, Oberwarth); success has been attained both with a relatively small addition of carbohydrate (10 gm. meal, 40 gm. cane sugar or food sugar), and also with a plentiful addition (15 gm. meal and 60 gm. sugar per litre), and also with "Valbel's preserved milk." The following table taken from a case of Birk's illustrates the size of the feeds:—

| | Amount drank | No. of meals | Body weight |
|---------|-----------------|-----------------|----------------|
| 1st day | 40 | 2 | 1,250 |
| 2nd " | 55 | 4 | 1,420 |
| 3rd " | 47 | 4 | 1,340 |
| 4th " | 55 | 6 | 1,230 |
| 5th " | 200 | 5 | 1,200 |
| 6th " | 140 | 5 | 1,200 |
| 7th " | 145 | 5 | 1,220 |
| 8th " | 175 | 5 | 1,240 |
| 9th " | 180 | 5 | 1,250 |
| 10th " | 200 | 5 | 1,220 |
| 11th " | 250 | 6 | 1,340 |
| 12th " | 275 | 6 | 1,480 |

Together with the nourishment of which the technical problems often present particularly great difficulties during the first days of life, it is the thermolability, especially hypothermia, which requires the greatest attention in the treatment of premature infants. As Pfäundler has said, the precautions which must be exercised consist, not so much in the actual administration of warmth (which, strictly speaking, is only possible when heat passes from the surroundings into the child's body, as when the surrounding temperature is higher than that of the child), but much more in measures to prevent excessive heat losses.

In order to attain this end it is chiefly necessary to take care that the post-natal fall of temperature is limited as far as possible. The methods of warming to be mentioned must invariably be applied in the very first hours of life. Budin has shown by statistics that the mortality of debilitated infants, under otherwise similar circumstances, is smaller when the initial cooling is limited. Doctor and midwife should therefore realize that progressive cooling should be prevented, not after the first bath and after the mother has been attended to, but that immediately after birth the child must at least be protected by being wrapped in warm clothes or laid between hot-water bottles. The recommendation of Polano to put premature infants immediately into a bath full of warm water (38°) is a suitable measure and is thoroughly justifiable. It is an iniquitous practice, and one of which the doctor should be warned, that of bringing a premature child one or two days old, merely wrapped in a cushion for its christening, into the open air before it has recovered from the initial loss of heat.

The precautions recommended for combating hypothermia are very numerous. Particularly great care and ingenuity have been applied to the construction of the so-called "couveuses." Since their invention by Tarnier in 1878 innumerable modifications and improvements have been submitted (for this subject consult Coerny-Keller). Of the more modern couveuses that of Finkelstein and Romel deserves mention. The couveuse consists of a closed chamber with warmed air; the principal requirements, which are more or less fulfilled by the newer models are, according to Pfäundler, the following:—

(1) The air reaching the child must be fresh, pure and sufficiently warm and moist.

(2) The temperature and water content of the air must be constant and possible to regulate.

(3) The couveuse must comply with the modern aseptic requirements of infant welfare—that is, it must be washable, able to be disinfected and easily cleaned, without unnecessary dust collectors, and awkward corners, &c.

(4) The couveuse must not greatly increase the work of the person in charge, who must always keep an eye on the child.

The small incubators, which were originally shaped like a cradle made of wood and closed above by a window, have been

superseded by large, more roomy models, the wooden walls have been replaced by glass, and the linings of the windows and also the whole framework are now made of metal or porcelain, or something similar and enamelled. Hot-water receptacles, permanent water baths, thermophore pillows, &c., serve as a source of heat; a constant source of warming is preferable (petroleum, gas, electricity). The problem of maintaining a constant temperature is well solved in the newer forms of couveuse by the automatic regulation of temperature. The technical difficulties regarding the moistening of the air are very considerable. Although in many of the old couveuses the air was too dry, it is, on the other hand, undesirable for the moisture to be too great; the prevention of the evaporation of water from the surface of the body which occurs in a space saturated with water vapour is not only extremely unpleasant, but is also not without danger. For this reason the "couveuse humide" of Bonnaire has not proved satisfactory. The difficulties of providing adequate ventilation are also considerable. This is best achieved by a suitable arrangement of the heating apparatus and the provision of a chimney. In fixed couveuses in institutions outdoor air may be used, but otherwise ordinary room air must naturally be used. A cotton-wool filter has been interposed in the air current to prevent contamination with dust or bacteria.

The most perfect type of couveuse is the warm chamber devised by Escherich and L. Pfandlér. This is a small chamber built into the room and closed on all sides and large enough to hold two infants; it is well lighted by windows and glass walls, and is provided with ventilation from the open air, gas heating which can be regulated, ventilation tubes and moistening apparatus. The "chambre couveuse" makes it possible to carry out the whole daily routine (feeding, bath, cleansing, &c.) in the equally warmed inner chamber of the room.

A yet further step has now been made, and instead of the couveuse described, a whole room has been fitted up to form an incubator which may be entered by a grown-up person. Such an incubator is essentially nothing more than a room which has been heated to a higher temperature (25° to 30° C.); it only remains for care to be taken that ventilation and humidity are sufficient. But it must certainly not be forgotten that it is very uncomfortable for the person in charge to have to remain in such an overheated room, and also that the cost of such an arrangement is very considerable (Pfandlér). None the less couveuse rooms have proved very useful in institutions, and should work out considerably cheaper than the very costly incubators which so frequently need repair.

The temperature of a couveuse should not, as a rule, exceed 25° to 26° C.; only with very small badly chilled infants should the temperature be raised at first to 28° to 30° . Higher temperatures (30° to 34°) used to be employed, but have now been abandoned.

It appeared that such temperatures were very liable to cause very injurious overheating of the child. Moreover, the warmed air reduced the respirations, since the stimulation to deeper respiration caused by cooler air was absent, so that the filling of the lungs was very apt to be interfered with (Kross, Meinert, Polano). With lower temperatures this fear seems unfounded; at least, Birk did not find the occurrence of attacks of asphyxia any more frequent in the couveuse room than in ordinary room temperature.

The opinions in respect of couveuse treatment of premature infants are very contradictory. The enthusiasm of earlier years has of late considerably cooled. It is undeniably true that the introduction of the couveuse has greatly assisted the care of premature infants. It is also probable that the couveuses which comply with the modern requirements of plenty of space, ventilation, moistness and light may not have any disadvantages for the child if the temperature is moderate; moreover, the opinion so frequently heard, that couveuse infants have little resistance to infection, is hardly founded on fact. It has also gradually been learnt that with suitable care the advantages of couveuse treatment may be obtained with simpler and cheaper materials. The author has for many years had the opportunity of observing the results obtained with Escherich's couveuse cells in St. Anna's Children's Hospital in Vienna, and was able to convince himself that they were not in proportion to the cost required. Even the previously mentioned couveuse rooms—simply rooms warmed to a higher temperature for premature infants—show a considerable simplification. Such a room, however, could only be started in an institution which always had a large number of premature infants under care. For this reason—even with the help of modern technical advances—we are driven back to the old principles of applying direct warmth to the child without much warming of the air, and heat loss is prevented by suitable warming arrangements.

Small premature infants should be first of all wrapped up in cotton-wool. The upper and lower halves of the trunk are wound round with a layer of cotton-wool, the legs are also covered with the same material, and the child is then clothed with a small shirt or woollen jacket, the whole is then covered with a swaddling cloth. The scalp is also swathed in cotton-wool and covered with a cap, so that the face is the only part of the body visible. Urine and stools are received on small pads of cotton-wool. The child thus swathed is then placed between hot-water bottles, so that a bottle lies on each side and one at the foot. Ordinary earthenware bottles like "white beer" bottles or mineral water bottles are used; these are closed with beer-bottle stoppers, the tightness of which must continually be attended to; a small bandage should be used as an extra precaution against leakage. Since with bottles lying horizontally the leakage of hot water cannot be entirely prevented, bottles shaped like a long flat roof are to be recommended, having the opening on top (fig. 27). The bottles are filled with hot, i.e., 70°

water and wrapped in cloths. They are changed one to three-hourly, and in such a way that in regular rotation only one of the three bottles is replaced by a fresh one. The temperature under the coverlet, which is spread out over child and bottles, may be up to 30° , and with badly chilled debilitated infants up to 32° to 34° , and may even be raised at times to 37° (Oberwarth).

In place of the usual hot-water bottles a U-shaped hot-water container may be used, which surrounds the child at the sides and feet (fig. 28). Also thermophore plates of metal and thermophore cushions may be used with advantage. Cramer recommends an electrical heat covering, an electrophore wrapped in a layer of wool,



FIG. 27.—Bottle-shaped hot-water bottle.



FIG. 28.—U-shaped hot-water bottle.



FIG. 29.—Heating bath tub for premature infants.

which may be attached to an electric lighting plug in the wall of the house; the temperature may be regulated at different heights with stops and kept constantly by interposing a rheostat. Dufour recommends that premature infants should be wrapped in rubber material to limit the heat loss; ventilation may be obtained by a few holes in the material. The method raises doubts on account of the ease with which interference with perspiration occurs.

Of great use also are the so-called double-walled bath-tubs, between which hot water may be run in (fig. 29). According to Oberwarth, such an apparatus may be improvised by putting two tubs of different sizes one inside the other; the larger of the two must have an outlet in order to make a four to six-hourly filling with warm water (40° to 50°) possible. The heat of the water and of

the inner space are controlled with thermometers, and the whole tub is covered with a woollen covering. Modern institutions have warming tubs in use which are heated by constantly circulating water; the temperature of the immediate surroundings of the infant amounts to 31° to 34° (Birk).

The continuous warm bath recommended by Winkel consists essentially in leaving the child permanently in warm water, which is maintained at constant temperature by the addition every one and a half hours to one hour, of half a litre of fresh hot water, and is completely renewed every six to eight hours; the tub is provided with a lid in which there is a hole for the head. This method has not been much used owing to the danger of infection from the bath water and the increased difficulty of the healing of the umbilicus. Repeated warm baths are none the less a very excellent method of combating hypothermia in premature infants.

All the different methods of combating the heat loss demand careful observation of the infant on the part of the nurse, whose attentiveness and care are of the greatest importance in determining the fate of a premature child. She must take care that the temperature of the body remains constant, that neither overheating nor hypothermia takes place; it is desirable that the body temperature should be maintained between 36° and 37° . In course of time this temperature may be attained with gradually diminishing application of heat from outside with two or only one hot-water bottle, and finally only with warm clothes. Infants usually remain only one to two weeks in a couveuse, very exceptionally longer. If a couveuse room be available, and in a private house a well-ventilated and then strongly heated room will serve for this purpose, then a much chilled infant can be placed in such a room for a few hours only, until the temperature has been to a certain extent rectified, it may then be brought into somewhat fresher air and kept warm with hot-water bottles alone. If a child has been in a couveuse several days or weeks, the temperature must be gradually reduced to about 20° before it is exposed to the ordinary room temperature.

In the treatment of premature infants a very important part is the stimulation of the defective respiration. Care must be taken to see that the respiratory excursions of the child are sufficient. This may best be done by reflex excitement of the respiratory centre, with stimulating the skin, or in the same way by causing it to cry, and thus to take deeper breaths. Should cyanosis occur similar measures to those employed in asphyxia after birth must be brought to bear—sharp slapping, artificial respiration (but not Schulze's method of swinging), and above all hot baths, and with addition of substances which stimulate the cutaneous circulation, such as ground mustard and sparkling oxygen baths (Schmid); if the fall in temperature be not too great a quick cold sponging or short immersion in cold water may be of use. Also the use of drugs which are able directly to stimulate the respiratory centre may be considered, e.g., ether, camphor, caffeine, and also atropine. Clinical experience on this point is still absent.

The inhalation of oxygen is very useful in these asphyxial attacks. Birk recommends that an oxygen cylinder should always be kept by the bed of a premature child, so that the funnel attached to the cylinder may be held in front of the child at any given moment. He thinks that in this way other manipulations for stimulating the respirations may be avoided in which there is danger of the food administered with so much difficulty being vomited up.

The fate of a premature infant depends on various circumstances. It is natural that the relative defective vitality due to the premature condition under otherwise similar circumstances will be greater the earlier the intra-uterine development has been interrupted. Finally, a limit of vitality is reached beyond which the organs are not in a condition to satisfy the demands of extra-uterine life. This limit is not sharply defined. As a rule the limit of vitality fixed by the age of the fetus falls in the sixth month of pregnancy. For this reason the prognosis is to a certain extent dependent on the absolute body weight. Premature infants weighing more than 2,000 gm. at birth have, as a rule, a very favourable prognosis in the absence of pathological changes, with infants under 1,000 gm. the prognosis is most doubtful. The smallest premature infants who have survived had a weight of 719 gm. (Rudmann), 750 gm. (L. F. Meyer, d'Ourepont, Oberwarth), 840 gm. (Maygrier and Schwab), 860 gm. (Birk, Pfandler), 935 gm. (Rodin), 950 gm. (Villemain), 960 gm. (Henbner), 980 gm. (Finkelstein), &c.

How greatly the vitality increases with the size of the body is shown by the following table from Ostrel concerning the fate of infants born with artificially induced labour:—

| Weight | Vitality | Length | Vitality |
|-----------|-------------|--------|--------------|
| 1,400 gm. | 0 per cent. | 40 cm. | 21 per cent. |
| 1,500 " | 0 " | 41 " | 20 " |
| 1,600 " | 17 " | 42 " | 25 " |
| 1,700 " | 27 " | 43 " | 28 " |
| 1,800 " | 21 " | 44 " | 51 " |
| 1,900 " | 35 " | 45 " | 50 " |
| 2,000 " | 47 " | 46 " | 55 " |
| 2,100 " | 50 " | 47 " | 58 " |
| 2,200 " | 43 " | | |
| 2,300 " | 49 " | | |
| 2,400 " | 38 " | | |
| 2,500 " | 54 " | | |
| 2,600 " | 60 " | | |
| 2,700 " | 50 " | | |
| 2,800 " | 65 " | | |

From these figures it may be seen that there is a very sudden rise in vitality about the weight of 2,000 gm. and the length of 44 cm. The statistics concerning the future of premature children born by artificially induced labour are in general very favourable (Rasellou, 89·4 per cent.; Heymann, 71·2 per cent.; Ahlfeld, 90·9 per cent.; Lorey, 71·7 per cent.; Hunziker, 83·5 per cent.; Ostrel, 56·9 per cent.).

The fatalities among premature infants occur, in the great

majority of cases, in the first days of life. Death occurs either because the organism is not sufficiently developed to meet the requirements of extra-uterine life, or from some constitutional weakness or definitely pathological cause. The determination of the cause of death is, moreover, not always possible on the post-mortem table. Undoubtedly many premature infants only die because they do not receive the necessary care during the first days of life. Spartan principles, evolved from the consideration that it is not worth while to permit the survival of the unfit, do not apply in any way to premature infants. If it is possible to keep a premature child alive through the dangers of the first period of life, provided that it comes from healthy parents, it may develop into a human being normal in every respect; this is a fact which cannot be sufficiently emphasized.

The chief clinical signs regarding the chance of survival of a premature child consist in its capability to react to stimuli. If a child be made to cry, or if it cries spontaneously, it can always be regarded as a favourable sign. The more pronounced the somnolence, the more defective the reflex excitability, the more doubtful is the prognosis. Such infants usually do not have a fresh complexion, they frequently become cyanosed, they have a tendency, in spite of the plentiful application of heat, to have subnormal temperatures, they frequently show sclerodermic changes in the skin. All these are signs of an unfavourable prognosis, which also show that an absolute form of debility is present. Such signs may be much more marked in infants of relatively high weight at birth than in infants of 1 to 1½ kg. For this reason for weights over 1,000 gm. the general appearance is of much greater prognostic importance than the absolute weight.

PART IV

Debilitas Vitæ, Constitutional Weakness, Diatheses, Constitutional Diseases

THE importance of the constitution has of recent years been much to the fore in medicine. One has learned to recognize how important individual tendencies are in determining the course of the most various diseases, how differently each individual acts in response to exactly identical stimuli, whether these be pathogenic or of a kind which may strictly be regarded as physiological. The paediatrist in particular has special opportunities of making such observations; different infants under his care and under the same methods of feeding sometimes will thrive wonderfully, and sometimes fall into a state of atrophy or die. Some of the children whose constitutions do not correspond to the requirements of the word "healthy" belong to the category which is usually classed under the ambiguous expression "defective vitality" or "debilitas vitæ," others become ill later with symptoms of the so-called diatheses.

Defective vitality is the designation for those conditions in which a child reacts to physiological stimulations with the symptoms of disease; by means of such stimulations, which are easily overcome by the healthy organism, its development is hindered and it is injured. This defective resistance and toleration may be overcome in the course of development; it may, however, reach such a degree that the organs are not able to meet the functional demands made on them, and therefore further life is impossible. No primary anatomical changes in the organs are present in these cases. True vital debility is solely a weakness of function. This functional weakness must at all events have its ætiological foundation in injuries inherited from the parents, these being due either to a specific disease of the latter (tuberculosis, syphilis) or to an unknown agent. In discussing the premature condition attention has already been called to its relation to defective vitality; and the differences between the relative defective vitality of the healthy premature child and absolute defective vitality have been pointed out. The two conceptions are independent of one another, but not infrequently they partially overlap. Pfäundler has distinguished six types or combinations:—

- (1) Debility on account of premature birth with an unhealthy mother.
- (2) Debility of mature infants of an unhealthy mother.
- (3) Debility on account of premature birth from a healthy mother.
- (4) Debility from other causes with healthy mother (twins, &c.).
- (5) Prematurity without debility.
- (6) Birth from unhealthy mother without debility.

Corresponding to the obscure ætiology and the absence of anatomically recognizable changes in the organs due to deficiency, the clinical symptoms of defective vitality are only very vaguely differentiated. They are generally cases of delicate children below normal weight, with low power of reaction, weakness in suckling, pale appearance and flabby condition of the skin. The diagnosis of defective vitality is often made in preference to others when a child dies during the first days of life without any characteristic symptoms of disease; by a careful post-mortem it is usually possible in such cases to show that a septic or toxic process, a cerebral hæmorrhage, &c., lies behind the "defective vitality." The diagnosis of "defective vitality" is only justified when no anatomical cause of death can be discovered at the post-mortem. If an infant declared to be of defective vitality survives, then the suspicion always arises that some disease has been present, which on account of the absence of obvious clinical signs, remained unrecognized. In spite of this justifiable limitation of the scope of defective vitality the condition none the less exists.

The debility may show itself as a lowered resistance towards disease, and such a condition can naturally not be identified in the post-mortem room. Stolte has called attention to the early death of numerous children in one family. The infants succumbed during or shortly after birth to injuries, which they met with during their fetal existence, in extra-uterine life or during birth. Stolte also attributes an ætiological importance to hereditary neuropathic tendencies. The children of such parents appear perfectly normal immediately after birth; in particular, outward signs of degeneration are often entirely absent, nevertheless they often show, even in the first days, signs which arouse the suspicions of a careful observer; twitching during sleep, nervousness, crying for no palpable reason during the whole night. Death takes place suddenly and unexpectedly after a little while, being preceded by symptoms of intestinal disease or convulsions. Stolte thinks that in such infants there is a congenital abnormality of the nervous system. He mentions a family in which five boys all died from convulsions in the first half month.

Every intermediate stage between such extremes of functional deficiency and the normal is represented. Thus, for instance, in such infants as those who, in spite of plentiful nourishment from the breast, do not develop normally and do not regain their birth weights for several weeks, a definite deficiency of organic functions must be assumed, even when there are no

symptoms of disease. If an infant reacts badly to the premature employment of artificial feeding, this condition must then be described as due to the presence of a relatively inadequate constitution. Such infants cannot be described as of anomalous constitution, since the artificial feeding cannot be described as physiological for the young infant. Also the symptoms of dyspepsia so frequently observed in breast-fed infants, when they persist after the first eight to ten days, must be attributed to an inborn, abnormal susceptibility of the intestinal mucous membrane. It is very important to realize this so as not to regard such disturbances as independent of the child, e.g., badly prepared or tainted food, inadequate mother's milk, &c. Many infants, during the first period



FIG. 32.—Newborn infant with myxodema. (Noted by H. Abelt.)

of life, exhibit an appearance which, to a certain extent, is like that of a premature child, without there being any shortening of pregnancy. They are usually smaller, more delicate, and weaker at sucking than the normal child. This perhaps is a case of intra-uterine inhibition of development. The limitation of the conception of the word "healthy" is very difficult. From the clinical point of view it must be made sufficiently wide to embrace infants of delicate constitution, including those with an especially susceptible intestine, although in other respects they do well.

The determination of the constitution during the newborn period can hardly be made with any certainty; if no pronounced symptoms of debility are present, we are unable to draw any conclusions from outward appearances as to the vitality of the child at the time.

The latency of the child's disposition is still more marked in the

so-called diatheses which play so important a part in the pathology of infancy and childhood; we are here dealing, not with diseases, but, as Pfäundler expresses it, with "tendencies to disease." Only the pathological tendency is congenital; active forces are yet necessary in order to produce pathological changes and the symptoms of disease. Even when these forces act in early infancy, a definite latent period is always interposed before the appearance of clinical symptoms. Czerny, to whom we are indebted for the formulation of the conception of the "exudative diatheses," points out that, even a history which suggests an inborn predisposition does not permit the diagnosis of a diathesis in the newborn child, and that a decision can only be arrived at, after weeks or months, as the result of definite dieting. Reliable means of recognizing the disposition are as yet unknown. Freund has described a tuft of hair as a characteristic early symptom of the exudative diathesis, which is already present in the newborn; in the parietal region the hair is directed anteriorly above and towards the middle of the head, there is thus a roof-like ridge corresponding to the middle line; when looked at from in front it is a triangular, gable-like tuft overhanging the forehead. Although this sign may be characteristic of the exudative diathesis, its absence does not permit of the conclusion that the child in question is necessarily free from the diathesis. Possibly certain appearances of the skin in the newborn are signs of an exudative skin tendency (*vide infra*).

It will be discussed later on to what extent lymphatism, spasmodophilia, and rickets are manifest in the newborn.

The practical conclusion which we must draw from the latency of the constitutional anomalies and diatheses is that by prophylactic measures we must prevent, as far as possible, the actual illness of a child which has perhaps a tendency towards the disease; although in many cases the anxiety may be unfounded, it is none the less best for the child if the hygienic and dietetic rules be rigidly adhered to from the first day onwards.

Only two of the true constitutional diseases which present a characteristic picture deserve mention, viz., myxedema and mongolism.

Although congenital myxedema depends on an inborn hypoplasia or aplasia of the thyroid gland, the characteristic somatic manifestations of the disease only appear during the first years of life. Probably the thyroid secretion of the mother enters vicariously either by the placenta or after birth by the milk. Nevertheless, in exceptional cases typical myxedematous changes are already present in the newborn. Abels has described a case in which thyreosplasia was proved by post-mortem examination; the child was born of a strumous mother (fig. 30).

The symptoms peculiar to mongolism are obvious even from the day of birth, and may usually be detected at a glance, particularly the broad bridge of the nose, the slit-like eyes, the well-marked epicanthus, also the enlargement of the tongue, the absence of



FIG. 31.—Mongoloid (one week old).

muscular tone, the looseness of the joints, &c. (fig. 31). Also the mental disturbance frequently associated with the mongoloid condition, like most conditions of idocy, becomes noticeable even during the first period of life by the striking quietness and indifference of the child.

PART V

Birth Injuries

BIRTH injuries are described as being solutions of continuity of the infantile body which are due to the action of the mechanical pressure of the act of birth on the foetus. The birth process even under normal conditions is to a certain extent to be considered as traumatic to the infant's body. A certain amount of pulling and pressure is always involved in the passage of the child through the genital tract. To these is added general venous congestion, either local or due to interruption of the placental circulation before the first inspiration, and which may cause the rupture of minute vessels. There are thus birth injuries which, on account of their occurrence in the normal birth process, may almost be described as physiological, like the caput succedaneum on the part of the body first born.

The trauma which gives rise to the birth injury may either originate from the genital passage of the mother, or it may be due to obstetrical manoeuvres (instrumental or manual). In artificial deliveries it is either the manual help in breech or transverse presentations (version and extraction) or the forceps which cause injuries to the child; in spontaneous delivery it is a case either of a disproportion between the size of the child and the pelvis of the mother, such as chiefly occurs in pelvic deformities, or of abnormal presentations. Birth injuries are found chiefly after prolonged labours, but may also occur when delivery has been particularly quick, if very powerful pains drive the child forcibly through the genital canal.

The injuries affect either the external soft parts, the skeleton, or the internal organs. They are most frequently found in the region of the head or the limbs, while the trunk is relatively rarely affected.

INJURIES OF THE EXTERNAL SOFT PARTS

(A) Injuries in the Region of the Skin and Subcutaneous Tissue

(1) CAPUT SUCCEDANEUM.

or swelling of the presenting part, occurs after the outflow of amniotic fluid, and is situated on the part of the body lying in contact with the os uteri. In consideration of the prevalence of

the occipital presentation, the caput succedaneum is most frequently found over the posterior portion of the anteriorly placed parietal, or on the back of the head. The caput succedaneum is formed thus: Owing to the close embrace of the os uteri and the soft parts of the pelvic diaphragm, the backflow of blood from the uncovered part is interfered with. Moreover, the difference between the atmospheric pressure and that of the uterine cavity plays a part in the causation of the swelling, because in this way a kind of suction acts on the presenting part. The swelling is derived from a serous infiltration of the tissue due to obstruction of the flow of lymph and blood, the latter causing the occurrence of numerous small hemorrhages (fig. 32). Extravasation of blood also takes place in the skin and subcutaneous tissue of the neighbourhood. According to Lönnberg, transudation takes place chiefly between



FIG. 32.—Frontal section of caput succedaneum over the right parietal. (After Böhm.)

the galea and the periosteum; widespread areas of hemorrhage sometimes are present under the periosteum; even the corresponding parts of the dura tend to be hyperæmic.

The situation of the caput succedaneum depends upon the presentation of the case in question. In the occipital presentation it is to be found on the posterior portion of the right parietal; in the second position, of the left; in the frontal presentation, in the region of the anterior fontanelle; in the face presentation, on the side of the face which has been against the anterior wall of the pelvis; in brow presentation, on the forehead; in the breech presentation the swelling is usually in the gluteal region, on the posterior and outer part of the thigh (it may, however, also affect the scrotum or penis, or the labia majora). In prolapse of the arm or leg, the skin of the presenting extremity shows the characteristics of a caput succedaneum; these consist of a soft, doughy, often very considerable swelling of the soft parts, a bluish coloration of the skin, which

usually also exhibits numerous minute extravasations. This is best seen when the swelling is localized in hairless parts of the body. The caput succedaneum is most disfiguring in face presentations, when lips, cheeks, nose and eyelids are often much swollen and redematous, showing bruises and a bluish-red coloration (fig. 33). Sometimes, in the neighbourhood of the swelling, bullæ are formed, which are usually about the size of a pea and filled with clear, yellow fluid.

The extent of the swelling tends to be about as much as can be covered by the palm of the hand, and is usually roundish. It frequently appears, especially when situated on the head, in the form of an elongated swelling, this being due to the fact that one zone of arrested circulation is succeeded by another.

The intensity of the swelling varies. It depends on the strength and duration of the pains from the rupture of the amnion until the end of the second stage of labour. As a rule, the stronger and the longer the pains the greater is the swelling. A second factor which plays a part in the formation of swellings is the strength of the constriction by the os and cervix of the uterus. In exceptional cases the constriction may be caused by a cervix which has undergone connective tissue degeneration. Spastic stricture of the upper part of the cervix may, even with weak pains, give rise to the formation of an extensive caput succedaneum (Ehrendorfer).

The caput succedaneum usually tends to disappear during the first twenty-four hours, or at least it becomes greatly reduced. An infiltration is practically never noticeable after two or three days. The only recognizable signs during the first week are the bluish coloration and the extravasations of blood, and these diminish in intensity. Later on these disappear entirely. A favourable prognosis may thus be given even in very disfiguring swellings in the face.

In exceptional cases, however, a part of the caput succedaneum may undergo a necrotic or gangrenous degeneration which may in places reach down to the bone. This is naturally not without danger for the child, and may even lead to fatal complications (Ehrendorfer).

In hæmorrhagic diseases it may happen that the caput does not diminish, but hæmorrhage from the torn vessels ensues, so that at the site of the swelling an extensive subcutaneous hæmatoma develops (*vide infra*).

In uncomplicated caput succedaneum treatment is superfluous. Complicated cases should be treated in accordance with the usual surgical principles.

(2) MARKS OR TRACES OF PRESSURE.

While the skull of the infant is passing through the pelvis it is subject to pressure. Thanks to the compressibility and malleability of the infantile skull, the head usually moulds itself in such a way that the excessive effects of pressure are obviated. Nevertheless, especially when there is great disproportion between



FIG. 25.—Caput succedaneum in facial presentation. (After a plaster cast in the Schaff-Prümers, Vienna.)

head and pelvis, e.g., in a narrow and flat pelvis, such a pressure may be brought to bear on the head when it is stuck in the pelvis or passing through it, that the projecting parts of the pelvis (particularly the promontory) leave marks behind them. In this way red patches or stripes arise, usually very typically localized; in their neighbourhood small extravasations of blood or bruises due to pressure may be noticed. A streak usually runs from the upper angle of the parietal bone, along the coronary suture, to the ear. It may go first from the anterior fontanelle towards the parietal eminence, and then course towards the temple, at an obtuse angle, or it may extend from here on to the face in the cheek region. In this way angular pressure marks arise; in place of streaks there are sometimes found in the same region individual areas separated by unbroken skin. Sometimes, parallel to the stripes described above, a second stripe is visible which is due to a false promontory, or it may be caused by an alteration in the position of the head at the pelvic inlet. With flat pelvis a second pressure mark is sometimes found on the opposite side on the forehead, the cheek, or the neighbourhood of the eye. This is due to the horizontal ramus of the pubis. With uniformly contracted pelvis, in which the pressure is more equally distributed, irregularly arranged pressure marks are to be found, usually very small, but more intense (Küstner). If part of the skin of the head be subjected to the pressure of the promontory for a longer time necrosis and ulceration may result; this gradually sloughs off. In such cases a scar sometimes results. By infection of an open wound an abscess may be formed, and even a widespread cellulitis (Shukowski). Usually, however, only a slight contusion is present, the last traces of which disappear during the first weeks. This also applies to superficial excoriations which are occasionally to be found.

Pressure marks occur almost entirely in head presentations; when the head is born last they are hardly ever visible. In other parts of the body they are also very rarely seen. Pressure marks on the arm are found in prolapse of the arm when the latter is compressed between the head and the pelvis. Should the jamming persist for a long time, necrotic and gangrenous processes may possibly follow.

On the neck marks due to stretching may be observed. They are found after spontaneous delivery in head presentations, and, in fact, on the side of the neck which has been stretched most. In occipital presentations they are on the dorsal, in face presentations on the ventral aspect of the neck. They consist partly of parallel, partly of reticular transversely directed stripes, which tend to fade away after two or three days (Kaltenbach).

(3) FURTHER INJURIES OF THE SUPERFICIAL SOFT PARTS.

After delivery with forceps pressure marks are very frequently found on the skull. Usually the regions most affected are those

against which the ends of the forceps lay; thus on a transversely gripped head they will be on the cheeks. If the head be gripped obliquely marked swelling of the eyelid will be found on one side and on the other a pressure mark near the ear, or horseshoe-shaped impressions in the region of the zygomatic arch and on the forehead, sometimes associated with facial paralysis. If the forceps be applied in the middle line of the head the impressions will be situated on the forehead and occiput. The marks of pressure caused by forceps consist either of irregular red blotches and stripes or of small subcutaneous hæmatomata. Sometimes hæmorrhagic infiltrations, about the size of a hazel nut, are formed symmetrically on both cheeks. On the forehead in the region of the scalp small linear wounds are sometimes seen, evidently due to tearing of the superficial layers of the skin.

Rough handling with the forceps (especially if they slip) may lead to deeper, subcutaneous injuries and wounds which leave scars behind them. Erskine observed a case in which the ear had been torn off by the slipping of the forceps. H. Schröder mentions a case in which the whole scalp was raised by blood flowing from the supra-orbital artery which had been torn by the end of the continually slipping forceps.

The prognosis of all these injuries naturally depends on the intensity of the force applied. The usual impressions made by the forceps disappear as a rule within the first weeks.

Carelessly conducted artificial delivery may give rise to very various injuries to the outer soft parts, whether it be by the finger or finger-nail of the accoucheur's hand or by the instruments. Thus, for instance, it has happened several times that the accoucheur, intending to rupture the amniotic sac, mistakes the presenting head for the membrane, and incises it with scissors, or some other sharp instrument (Adamkiewicz, Dietrich, Kratzer, and Dorf).

The application of Veit-Smellie's method, in which the finger is introduced into the mouth when the head is born last, may, besides causing injuries to the tongue, floor of the mouth, and hard palate, also tear the frenulum lingue and the angle of the mouth. Strassmann has observed a case of fatal hæmorrhage from a torn frenulum.

Injuries to the lower end of the trunk are not infrequently met with in careless examinations or operations in cases of breech presentation. The injuries affect the anus (the foetal anus being mistaken for the os uteri), the perineum, scrotum, and vagina. Contusions, excoriations, and deeper wounds of the lower limbs, especially of the iliopectineal fossa and of the genitalia may be caused in delivery by the finger or by obstetrical instruments (noose, breech hook). Meurer describes a case of perforation of a hydræcele mistaken for the amniotic sac. In version of the foetus, strong pulling of the foot may result in tearing of the perineum (Stumpf).

Amongst the consequences of birth we must finally mention ecchymoses, which may be found in the scalp region, on the fore-

lrad, the conjunctiva, and sometimes on the buccal mucous membrane or retina. Strictly speaking these are not birth injuries, but the result of venous congestion which causes the rupture of small blood vessels.

Injuries to the soft parts (skin injuries and hæmorrhages) may also, though rarely, occur within the uterus, where there is trauma of that organ. In medico-legal literature several such cases are recorded (Höfmann, Kolisko).

(B) Injuries affecting Muscles (Congenital wry-neck)

In both spontaneous and artificial delivery injuries to muscles are rendered possible. By tearing and stretching, muscle-fibres may be ruptured to a greater or lesser degree. In this way, intramuscular hæmorrhages may occur, and be detected partly clinically and partly anatomically. Injuries are especially likely to occur in such muscles as are in a contracted condition when affected by the traumatic agency (Mikulicz). With premature respiratory movements, the muscles of respiration run considerable risks (Kader). Köster found, in a child delivered by breech presentation, hæmatoma of the sternomastoid, the scalenes, pectoralis major, and splenius muscles. Hæmatomata of the sternomastoid are the most important and have been the most carefully studied.

Injury to the sternomastoid can take place in both head and breech-presentations; indeed not only in forceps and manual-delivery but also where birth is spontaneous. The hæmatomata are found in greatest relative frequency in breech-presentations. They occur when the shoulder is in the sagittal diameter of the pelvis while the head is placed in the same diameter of the pelvic inlet, or in head-presentations in which the head is already rotated in the pelvic floor, and the shoulders are still in a sagittal plane. In both cases the head has undergone a right-angled turn from the body (Schauta).

The sternomastoid which corresponds to that side towards which the face is turned, undergoes maximal stretching and may thus be easily injured. In view of this mode of origin, hæmatoma of the sternomastoid is generally unilateral. Bilateral hæmatomata have, nevertheless, been described. Hildebrand gives the following explanation: When in order to free the arm, there is very powerful twisting of the shoulder at a time when the head is immovable, with its long diameter in the sagittal diameter of the pelvis, this manipulation may, under certain circumstances, injure both sternomastoids.

Hæmatoma of the sternomastoid appears sometimes as a soft, sometimes as a solid, circumscribed swelling of a size between that of a hazelnut and that of a pigeon's egg, usually in the sternal part of the muscle. Not infrequently the surrounding soft parts, skin and subcutaneous tissue, are swollen and oedematous, so that the muscular tumour itself is not clearly palpable for some days. It moves with the muscle and does not seem particularly painful.

The most striking symptom is the oblique posture of the head, which is usually turned toward the healthy side, but the opposite may be the case. It must, however, be pointed out that this oblique posture of the head is by no means a constant symptom. Not infrequently the head is in quite a normal position.

Swelling and wry-neck may disappear after a few weeks or months without leaving any noticeable trace. After the rupture of the muscle, simple scar formation follows, which does not as a rule involve any shortening of the muscle. That the so-called *caput obstipum* musc. congenitum is a result of the hematoma of the sternomastoid caused by birth, has been disproved by the fairly numerous microscopical observations now available, and showing the absence of blood pigment. The *caput obstipum* congenitum has, by most authorities, been attributed to myositis fibrosa, in Mikulicz's interpretation of the term. Mikulicz himself believes that this myositis is, in the majority of cases, due to a contusion of the muscle against the symphysis or ligamentum arcuatum during delivery. Kader is of opinion that a traumatic infective myositis develops, due to an infection of haematogenous or intestinal origin. He thus believes that it is due after all to birth-injury. Most authorities, however, believe that the muscular changes have already begun in utero (Koster, Miloš, Pincus, Kehrer, Rettig). The aetiology of these intrauterine changes in the sternomastoid described as fibro-myositis, dystrophy, or developmental disturbances, is certainly not yet explained. The assumption of an infection taking place before birth is not a satisfactorily sound basis. Syphilis has also been suggested (Durante). Adhesions between the foetus and amnion are believed by Petersen to be the origin of congenital wry-neck, but this is improbable owing to the absence of external changes. Völcker describes *caput obstip. cong.* as an intrauterine, hereditary deformity: When the amount of amniotic fluid is not sufficient, the elastic pressure of the uterine walls causes lateral flexion of the head. In this way the shoulder is placed in a cavity formed between mandible and clavicle in the middle of which the vessels are situated, and are firmly compressed by the shoulder against the middle of the vertebral column. Should this pressure persist, the supply of arterial blood is interfered with, and venous congestion takes place. In this way a slow, fibrous degeneration after an originally spindle-shaped swelling takes place below the middle of the muscle. Finally, the sternomastoid on one side of the neck is represented by a shortened, hard strand; the head is bent sideways, and, as a rule, its perpendicular axis is somewhat displaced. Koster suggests that, on account of wry-neck, the foetus does not fit well into the genital canal, so that, in these cases, the *caput obstipum* may not be the result but the cause of a breech presentation. From the above, we may conclude that in the sternomastoid of the newborn there are two kinds of changes: (1) ruptures and hemorrhages caused by birth-injuries, which usually heal with simple scar-formation,

not interfering with function, nor tending to lead to permanent wry-neck; (2) changes beginning *in utero*, therefore congenital, and probably independent of birth-injury, which give rise to the condition of wry-neck.

The treatment of muscular injuries is limited to cool compresses and gentle massage. Should the head be held obliquely, it must frequently be righted and pushed toward the opposite side. Surgical interference is only indicated in true *caput obstipum* and should not, in any case, be employed during the first few months.

Hofstücker describes a hæmatoma of the masseter as a typical birth-injury. It is observed in manual delivery by Veit-Smolle's method. The cause may be the overstretching of the muscle in attempted rotation of the head by pulling back the laterally-rotated chin. The hæmatoma appears as a swelling of the cheek, about the size of a hazelnut, which becomes more marked during the first three days, but which afterwards diminishes. Its consistency is fairly solid, and the shape elliptic but not well differentiated. Sucking is not affected by the hæmatoma. The differential diagnosis of this condition is necessary, chiefly, with inflammatory conditions of the parotid gland. The absence of inflammatory symptoms, freedom of the region before and behind the ear, tendency to retrogression, and finally, the anamnesis, are of value in the diagnosis of masseteric hæmatoma. The condition probably clears up without trace. Treatment for the first days consists of cold compresses; later, gentle massage, from both within and without the buccal cavity, is substituted.

(C) Subperiosteal Hæmorrhage (Cephalhæmatoma)

Even after easy deliveries small and large extravasations of blood may be found in the tissues below the galea and particularly between the periosteum and the bone in the neighbourhood of the coronal suture. Should a large subperiosteal hæmatoma be formed, in which the periosteum is detached and raised from the bone, it is then described as a cephalhæmatoma. These cephalhæmatomata are usually found in the region of the parietal bones, less frequently over the occipital, frontal and temporal bones and, in exceptional cases, over the jaws. In the great majority of cases only one hæmatoma is present, which is usually situated over the parietal bone which is anterior during labour (fig. 34). The most frequent situation for a cephalhæmatoma is certainly over the right parietal bone. It is not very rare, however, to find two hæmatomata, usually over both parietals or over one parietal and the *planum occipitale*. Even three hæmatomata may occur. In these cases the head may assume most curious shapes. The size of the swellings is very variable. There are hæmatomata hardly as large as a nut and others as large as the infant's fist, which cause considerable deformity. They are hemispherical in shape (more rarely oblong), and are sharply defined swellings and never cross the sutures (this being characteristic of their subperiosteal

position) because although the periosteum is but loosely attached over the bones it is firmly attached to the sutures.

During the first two days the skin over a cephalhematoma is usually oedematous, a caput succedaneum being situated over it. It is not until this has subsided that the hematoma becomes definitely recognizable as such. Owing to continued hemorrhages from the ruptured vessels it tends to increase in size in the first few days, sometimes to the end of the first week. The originally

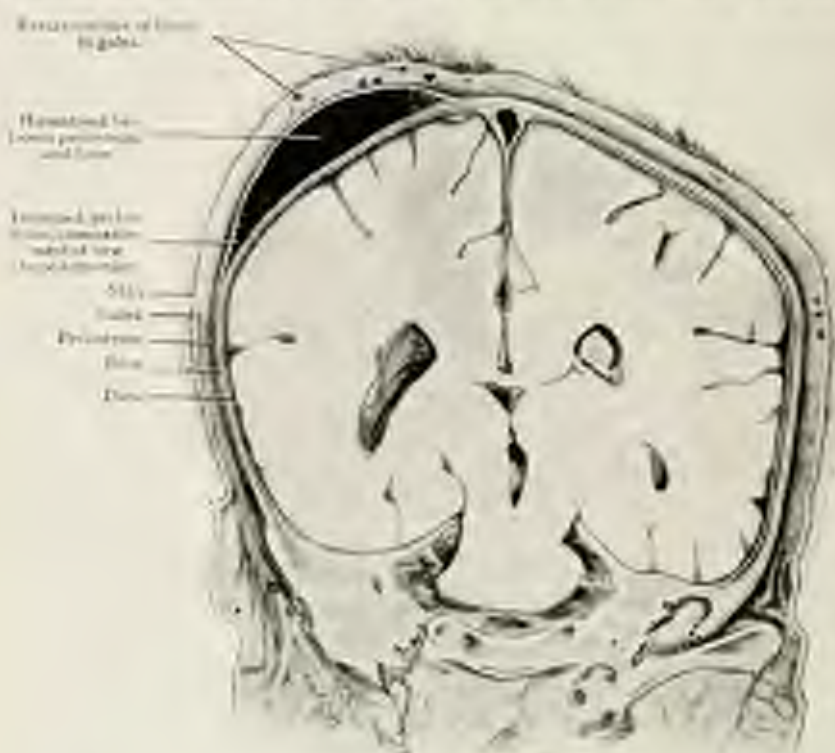


FIG. 34.—Frontal section of a skull with cephalhematoma on the right parietal bone. (After Donn.)

somewhat doughy swelling then shows definite fluctuation, since the coagulated blood gradually liquefies. The tumour has at this time the consistency of a tightly-stretched cyst. In the course of the second and third weeks, sometimes even earlier, a fresh symptom appears which is extraordinarily characteristic of the cephalhematoma. At its periphery a wall of bone is formed which becomes progressively more palpable; it is due to the activity of osteoblasts which form new bone at the junction of the dislodged and adherent periosteum. The formation of bone may sometimes begin from the detached part of the periosteum, so that the cephalhematoma

is then covered by a thin layer of bone, which on palpation gives a sound of crackling parchment. This, however, is not of frequent occurrence. The skin over the cephalhæmatoma does not often show any striking change. Sometimes minute extravasations may be found, or a diffuse, bluish-red coloration. Pressure marks may also be found in the same region. No considerable tenderness on pressure ever seems to be present.

From the second week onwards, the tension tends to slacken and a gradual diminution of the hæmatoma follows, owing to the absorption of the latter. The time taken for the resolution of the tumour depends upon its size. While small hæmatomata may have disappeared by the third or fourth week, larger ones take from six to eight weeks, and sometimes even three months to disappear entirely. As a rule cephalhæmatoma disappear without leaving any traces. Sometimes the bony wall, and a certain roughness of the bone within it, persist for a longer time. In rare cases, when the roof of the hæmatoma forms large amounts of bone, a more or less distinct eminence of solid bone remains, which resembles the original hæmatoma in position and shape.

The cephalhæmatoma probably arises as follows: the repeated backward and forward movement of the head by the pains loosens the scalp, which is approximated to the sides of the genital canal, and its connection with the bone is broken down. In this way multiple ruptures of vessels are brought about, from which the blood flows under the loosened periosteum. When several cephalhæmatomata are present we must assume that it is not only the soft parts which embrace the presenting parts, but also other parts of the canal to which the scalp is fixed in one or more places. The cephalhæmatoma is exceedingly rare in greatly contracted pelves, because here periodic back and forward movements of the scalp are limited or prevented (Lorand). As with all hæmorrhages occurring in labour, obstructed circulation and the ease with which vessels rupture are important accessory factors. In a few cases, small injuries to the skull bones (fractures and fissures) are also found. In cases of defective ossification of the skull bones a larger vessel may be injured, and lifting of the periosteum may be caused by the high pressure of the blood flowing from it.

In general, cephalhæmatomata are more frequent in the children of primipare, especially of elderly primipare, with rigid soft parts, after powerful pains. They are thus more frequent after quick than after protracted delivery. Not infrequently the forceps cause the scalp to be pushed away from the bone. The relative frequency of bilateral hæmatomata is greater when delivery is by forceps. In breech-presentations, cephalhæmatomata are exceedingly rare; a few such cases have nevertheless been observed (Stumpf). Cephalhæmatomata are noticeably more frequent in boys than in girls (2 to 1). According to the large statistics of Beck, there is one case of cephalhæmatoma to every 200 births (0.5 per cent.). According to Hennik, the frequency of cephalhæmatomata is 0.45 per cent.;

according to Hofnagel 0.6 per cent.; according to S. Meyer 0.5 per cent.

If there be a defect in the skull, whether a congenital gap or fissure or a fracture, the hæmorrhage may then pass to the inside of the skull and spread out between the dura mater and the bone. These epidural hæmatomata are described as internal cephalhæmatomata. These but rarely reach a considerable size and compared with other intra-cranial hæmatomata are only of secondary importance. The diagnosis can only be made when cerebral symptoms are present. These symptoms tend to be absent, however, owing to the smallness of the extravasation.

Of greater clinical importance is the suppuration of a cephalhæmatoma. The infection usually arises from small unnoticeable excoriations. As a matter of fact the mode of entry of the infection is often no longer recognizable when suppuration begins. The diagnosis of suppuration is easy when the outward appearances of inflammation are present, such as high temperature, redness, and swelling of the skin. This, however, is by no means always very pronounced. Fever also is not a necessary accompaniment. Suppurating hæmatomata certainly exist, in which the skin appears to be perfectly intact, and without fever. The suspicion of suppuration therefore only arises if after the first week the swelling not only fails to diminish but perhaps increases, when the turgidity persists and the fluctuation becomes more marked. The infants also tend to show pain when examined. In such cases it is better to ascertain the nature of the case by means of an incision.

The diagnosis of a cephalhæmatoma is usually quite easy. The roundish, sharply defined, fluctuating tumour, which does not cross over the sutures can hardly be mistaken for any other condition. The caput succedaneum has disappeared or at least diminished at the time when the hæmatoma appears. Only during the first two days it is sometimes difficult to decide whether a cephalhæmatoma is hidden under a caput succedaneum. Meningeoles show respiratory movements and pulsations, moreover they may be reduced and have a different position.

Therapeutic measures are usually superfluous in cephalhæmatoma. To protect it against pressure and injury a light pad of cotton wool may be placed on it. Only with severe extravasations without tendency to absorption is evacuation, by means of an incision, indicated. Suppurating hæmatomata must naturally be emptied by means of an incision.

(D) Injuries to Nerves

(1) OBSTETRICAL PARALYSIS

(A) OBSTETRICAL PARALYSIS OF THE UPPER EXTREMITIES.

Injuries of the peripheral nerves which take place during delivery occur, almost exclusively, in the brachial plexus and the facial nerve.

A paralysis due to an injury of the brachial plexus during delivery is known as obstetrical paralysis.

Obstetrical paralyses are almost always results of difficult and often of artificial deliveries. Out of ninety-four cases in the literature of the subject Stransky found the following birth history:—

- 31 cases in which forceps were used.
- 50 cases of manual assistance (extraction, freeing of arm).
- 27 cases of difficult, very protracted delivery.
- 11 cases of asphyxia.
- 2 cases of spontaneous delivery.

Stransky considered the increase in the venous characteristics of the blood caused by the difficult delivery to be an important factor in the aetiology of the paralysis. On account of the toxic qualities of the blood, as is well-known to the neurologist, the resistance of the peripheral nerves is reduced so that more or less harmless mechanical injuries may produce serious consequences. Obstetrical paralyses are found after all varieties of presentation. While Kennedy does not attribute to the presentation any essential influence on the kind of paralysis, other authorities are of opinion that the true Duchenne-Erb paralysis is found exclusively or, at least, almost exclusively, in presentations in which the head is born last (Peters, Jolly, Schüller).

With regard to the mechanical origins of obstetrical paralyses there are essentially two factors which may lead to a lesion of the brachial plexus: pressure (injuries due to compression) and strain (injuries due to pulling and tearing).

Direct pressure of the accoucheur's hand while delivering the head may affect the plexus. Prager's, Von-Smollie's, and Mauriceau's methods in which the index and middle finger of one hand are passed over the nape of the neck, are the methods in question; they may exercise pressure on the suprascapular fossa during the extraction of the head. It is at all events very unusual for the direct pressure of the finger of the accoucheur to be so powerful as to cause an injury leading to permanent paralysis. Such a pressure will even be but rarely exercised when the shoulders are delivered with upwardly directed arms.

According to Stolper paralysis by direct pressure of one blade of the forceps is only possible when the head is in an oblique position, when the rules of applying the forceps are not observed or when the deflection is not noticed. In rare cases pressure on the plexus may be caused by a hernatoma (Fritsch, Seeligmüller) or by the umbilical cord (Roulland).

Of greater importance is the pressure on the plexus by the clavicle. According to Schulze this may take place when an arm is thrown back over the head, in occipital presentations, when during the rotation of the head in the pelvic outlet the face is turned away from the shoulder, which is posteriorly situated at the time (Watson). Schoemaker explains the occurrence of compression as follows: The shoulder is pressed upwards or upwards and

inwards; in the first case contusion takes place between the clavicle and the first rib, in the latter case between clavicle and vertebral column. According to Stolper birth paralysis due to pressure of the clavicle occurs, especially when one arm (and the clavicle) is pushed strongly upwards and backwards.

Of greater importance in the genesis of birth paralyses than the pressure brought to bear on the plexus is the injury to the nerves caused by tension. If in head presentations the shoulder is pressed forcibly downwards and the head bent towards the opposite side, then part of the brachial plexus is stretched. By strong pulling of the head to facilitate the delivery of the shoulders nerve trunks may be stretched, pulled, or even torn (Carter, Stolper, Rühle). A more powerful pull on the head is provoked by narrowness of the pelvis and breadth of the shoulders. Of the greatest importance in this connection is the considerable lateral deflexion of the head towards the shoulder (Eversmann). Fieus was also able to show in experiments with animals that the essential factor is an oblique "asynclitic" pull, too strong a pull when the head is inclined towards the shoulder. In such a pull on the laterally flexed head, chiefly the fifth and sixth and, hardly ever, the seventh and eighth cervical roots are stretched. These results are in accordance with those arrived at in artificial delivery, which show that in typical cases it is the trunk formed by the union of the roots of the fifth and sixth cervical nerves, which is chiefly the site of the lesion. Stretching of the brachial plexus in head presentations may occur just as well as in breech presentations. When the head is born last, freeing of the arm, the pull on the shoulder and the pull on the arm are particularly dangerous. Schüller describes two cases of birth paralysis which were combined with *caput obstipum* of intra-uterine origin. He considers wry-neck to be a predisposing factor. Since, as has been proved experimentally, a 30° flexion of the head is sufficient to cause an injurious dragging on the plexus, in such cases the straightening of the head, necessary for delivery, is enough to cause serious stretching of the plexus.

The question as to what definite pathological lesions of the plexus are generally responsible for birth paralysis is a difficult one to answer, as so few recent cases reach the post-mortem room. Up to now our knowledge of this subject is derived on one side from infants who have died from some other disease, and on the other from operations in which the brachial plexus is dissected out. The anatomical findings are often negative. It must be considered as proved that conduction may be broken by mere overstretching of the nerve fibres without even any damage to nerve (Spitz). Often individual nerve branches are injected (Dauchez), or there is an extravasation of blood in the neighbourhood of the plexus (Danyau, Fritsch, Seeligmüller). In other cases degenerative or neuritic changes are found in the nerves (Oppenheim, Stransky). The most important, or at least the most striking, findings are tears of the brachial plexus or scars and connective tissue proliferations which

indicate a previous break in continuity (Eversmann, Kennedy, Spitzzy).

The lesion of the plexus sometimes affects the roots more and sometimes the parts of the actual plexus. They can hardly be distinguished clinically, particularly in the newborn, in which any disturbances in sensation due to lesions of the roots cannot be detected.

The existence of a paralysis is usually noticed a short time after birth. The newborn child already shows the peculiar tendency of the older infant of throwing the arms upwards and bending them at the elbow. Usually the first time the child is bathed and dressed it will be seen that one arm hangs down in a flaccid condition or swings to and fro.

Different types of paralysis result according to the situation of the injury to the plexus:—

(1) *Paralysis of the upper part of the Plexus (Duchenne-Erb Type).—*This represents the typical form of birth paralysis. In these cases the injury affects the fifth and sixth cervical roots or the trunk formed by their union. The latter contains bundles for the musculo-cutaneous nerve which supplies the biceps and brachialis anticus, for the circumflex nerve (deltoid muscle) and the musculo-spiral nerve (supinator longus and supinator brevis). Since the suprascapular nerve leaves the plexus at the same point the infraspinatus muscle which this nerve supplies is also affected. The paralysis of the deltoid prevents the abduction of the arm from the shoulder, that of the infraspinatus prevents outward rotation; the paralysis of the biceps supinator longus prevents flexion at the elbow and that of the supinator brevis supination of the forearm. The following condition results from the paralysis of these muscles: the shoulder is somewhat depressed usually in a forward and downward direction, a condition which is, however, not always clearly recognizable during the first days; the upper arm lies limp against the trunk and is inwardly rotated and adducted; the forearm is usually found in a slightly flexed condition and is strongly pronated, the palm of the hand is thus turned backwards or even towards the side; the wrist and the finger-joints are usually somewhat flexed, the thumbs being pressed inwards. If the attempt be made to cause movement by cutaneous stimulation the result will vary according to the degree of the injury, and the upper arm will either move slightly or not at all; supination will be impossible. The hand and the fingers are usually more movable or are entirely unaffected. In rare cases besides the nerves mentioned the subscapular nerve (for the *teres major* and *latissimus dorsi*) and the long thoracic nerves (for the *serratus anticus*) are affected sympathetically. The chief results of this condition are alterations in the position of the scapula.

The following type of paralysis may be looked on as the atypical form of obstetrical paralysis:—

(2) *Injury to the lower part of the Plexus (Klumpke's Type).—*This takes place when the lesion affects the eighth cervical and first thoracic nerves or the trunk formed by their union. From these nerve roots the median and ulnar nerves derive fibres. For this reason the paralysis affects the small muscles of the hand supplied by these nerves and also part of the musculature of the forearm—namely, the long flexors of the hand and fingers. Should the second root also be injured, and this is not uncommon in injuries of the lower part of the plexus, then the muscles supplied by the musculospiral nerve are also affected, the extensors of the hand and fingers. The symptoms of paralysis thus affects the movements of the hand and fingers, changes in the position of which are dependent on the inclusion of the musculospiral nerve in the injury, whereas, at least in uncomplicated cases, the movement of the elbow and shoulder-joints is unaffected. Highly characteristic accompaniments of injuries to the lower part of the plexus are the orulo-pupillary sympathetic symptoms, as the injured first thoracic root contains fibres belonging to the sympathetic. Narrowing of the palpebral fissure and myosis of the pupil with the preservation of the light reaction then occurs from paralysis of the orbitalis and dilator pupillæ muscles. Besides these there are usually distinct disturbances in sensation in the region supplied by the ulnar nerve; these, however, cannot be detected with any certainty in the newborn. The injury is evidently much more widespread than the paralysis of the upper part of the plexus, so that the sensory fibres which in large nerves are less easily injured than the motor may be directly affected.

(3) *Complete paralysis of the plexus*, in which all the muscles of the arm and forearm are paralysed, is very rare. In all cases which have been recorded they were complicated with serious lesions of the bones.

(4) *Combinations and mixtures of paralysis of the upper and lower parts of the plexus*, transitions from partial to total paralysis have several times been observed. Bruns often found the musculospiral nerve to be involved in injuries of the upper part of the plexus, in one case of injury of the lower part of the plexus there was an isolated paralysis of the infraspinatus. Erb noticed a combination of injury of the upper part of the plexus with median paralysis. In this way the most manifold types occur in which the two first-mentioned are merged.

(5) *Radiimentary Types.*—Sometimes paralysis of single muscles are observed, such as the deltoid (Bollenhagen) and the supinator longus (Oppenheim).

(6) *Bilateral paralysis* are very rare. Jolly describes a case of symmetrical bilateral paralysis of the pectoralis major, latissimus dorsi, triceps and almost the whole musculature of the forearm and hand. On account of the action of antagonists the arms assumed a hooked appearance. Bruns, who observed a very similar case, is of

opinion that in such bilateral paralyses the spinal cord is usually affected.¹

The visible symptoms of paralysis in the first days hardly permit of any conclusion as to the degree of the lesion which has affected the plexus. The progress of the condition naturally depends largely on whether the nerve trunks are merely stretched or whether they are torn, and also whether the injurious influence has acted a long or a short time on the plexus. Frequently there is complete paralysis in the first days, which, however, in the majority of cases becomes limited to its typical distribution during the first week, or it may subside still more and resolve itself into a paralysis of individual muscles. In favourable cases of Erb's paralysis the forearm begins to be more movable after a few weeks, and the improvement gradually continues proximally. In injuries to the lower parts of the plexus the course of recovery is reversed and proceeds distally.

Even in favourable cases complete recovery during the first two to three months can hardly be expected, but this may none the less take place after six months to a year. Should signs of paralysis still be present, then spontaneous recovery is no longer probable. Partial recovery may occur; one group of muscles will recover completely, while permanent paralysis affects another.

The prognosis, not unfavourable in general, must always at first be considered as doubtful. Stransky characterizes it as "*dubia in bonam vergens*." The results of electrical tests during the early stages of the disease must not be relied on too much. It is not until some months later that they can be used for prognosis. In a case of Schüller's the reaction of degeneration was given in the seventh week, in the ninth week the electrical excitability was quite lost. Sachs sums up as follows: Should faradic excitability be present, then even in severe cases recovery may be expected in two to three months. If only galvanic excitability be present, then the time is estimated at about six months. If both faradic and galvanic excitability be affected, then recovery, if it takes place at all, will not do so before one or two years.

At all events it must not be forgotten that after severe lesions of the plexus, a progressive atrophy of the paralysed muscles may develop with complete impairment of function of the arm in question. Of definite importance for the prognosis of the case is the presence of complications, especially of bone injuries, which are commonly associated with severe nerve injuries. The condition of the child after birth is also important, since in Stransky's opinion

¹ NOTE IN CORRECTION OF PROOFS.—Kautschke, who describes a similar case of symmetrical paralysis of both upper extremities, believes that such injuries are due to lesions of the nerves which are caused by the powerful backward movement of the head in face presentations. He points out that face presentations are as a whole relatively frequently associated with spontaneous paralysis. ("Ueber den Zusammenhang von Gesichtslage und spontaner infantiler Geburtslähmung," *Monatsschr. f. Kinderheilk.*, 11, Orig., 455, 1912.)

the longer and the more severe the asphyxia of the child after birth the worse is the prognosis. The paralyses due to pressure have usually a more favourable prognosis than those caused by stretching. Suitable treatment has considerable influence on the prognosis.

From the prophylactic point of view we must mention that, in obstetrical manœuvres, a strong pull must be avoided as far as possible, since it may lead to tearing of the brachial plexus; and we must similarly avoid any considerable flexion of the head.

If a newborn child be suffering from a birth palsy, the paralysed limb must be left at rest during the first days. Paritz recommends at this time dry compresses in the supraclavicular region. After one or two weeks light massage, warm baths and progressive movement may be introduced, and after four weeks (not later) electrical treatment. Faradic stimulation is given every day, or every other day, for three, five, or ten minutes. The electrical treatment must be continued with the greatest perseverance and energy if success is to be gained. Daily massage is given at the same time. Rohde recommends the healthy arm to be bound to the side of the body with bandages, so that all motor impulses will affect the paralysed arm.

For all cases with no tendency to heal nerve suture is the best remedy (Kennedy, Campbell and Spitzzy). In view of possible spontaneous improvement or of cure under electrical treatment, operation should be deferred for a time—at least until the end of the second month. Too long a delay has the disadvantage that the improvement, expected after the operation, tends to be delayed. Kennedy has compiled the following table:—

| Time after lesion. | | | | Time at which beginning of improvement is anticipated |
|--------------------|---|---|---|---|
| 1 month | — | — | — | 3 weeks after operation. |
| 2 months | — | — | — | 3 months after operation. |
| 3 " " | — | — | — | 3½—4 months after operation. |
| 3—6 " " | — | — | — | 7—8 " " |

The best time for operation is thus about the second quarter of the year. If, on account of the child's general condition at this time, operation be not advisable, it may be delayed until about the end of the first year. The operation consists in exposing the plexus, removal of any adherent scar tissue present, or uniting the ends of the nerve by suture after the excision of the scars. If the union of one nerve cord be impossible, anastomosis must then be considered between two different nerves, the injured one and a healthy one. If no scar tissue be present, an early operation may reinnervate the functional condition of one intact trunk (partial central implantation), or, in an older case, the total peripheral implantation of the injured trunk into an intact one, after prolonged and careful analysis of the paralytic symptoms (Spitzzy). Mikulicz

observed favourable results after stretching of the plexus in a five-weeks-old child.

All the operations mentioned on nerves, which appear so promising, are, naturally, only of use when the musculature has not atrophied nor undergone fatty degeneration. Should atrophy and secondary contractures once set in, only surgical orthopaedic treatment, such as tendon transplantation, need be considered.

The diagnosis of birth paralysis is not, as a rule, difficult, but in individual cases we can only arrive at the differential diagnosis after much consideration.

Above all, it is injuries in the region of the shoulder (dislocation) and the upper end of the humerus (fracture, separation of epiphysis) which may show symptoms very similar to those of birth paralysis. The correct diagnosis of bone injuries may be ascertained by means of X-rays and by the presence of crepitation. It must, however, not be forgotten that birth palsies may be combined with bone injuries. In dislocations the muscles are excitable and the shoulder-joint is sometimes tender on pressure. In rare cases cerebral paralyzes may be limited to one of the upper extremities, but, in such paralyzes, there are usually symptoms of motor irritation, convulsions, hypertonus and increased reflexes. Spinal paralyzes in injuries to the cervical cord and hæmorrhages in the vertebral canal are usually bilateral; in this class also bulbar symptoms are rarely absent. The differential diagnosis from poliomyelitis, which has even been described in young infants, may be very difficult, but we must remember that poliomyelitis in the newborn is exceedingly rare, and that a most exceptional case must be present if it only leads to a monoplegia. Syphilitic pseudoparalysis bears a certain resemblance to birth palsy. Osteochondritic swellings of the lower end of the humerus must be looked for, painfulness of this spot and swelling of the cubital glands.

(B) OBSTETRICAL PARALYSIS OF THE LOWER EXTREMITIES.

Paralyzes of the lower extremities analogous to obstetrical paralyzes of the upper limbs, due to stretching of the lumbar plexus, are very rare. Varior and Bonniot described a case of paraplegia of the lower extremities existing since birth, which, in view of the absence of sphincter paralysis, was attributable to a paralysis of the spinal roots in the neighbourhood of the second to fourth lumbar and of the first and second sacral segments, also to an excessive stretching during delivery. In differential diagnosis between this condition and that of extramedullary hæmorrhage within the vertebral canal involving the roots very great difficulties are encountered. Oppenheim mentions paralysis of the anterior crural nerve in a breech presentation; Bernhardt records paralysis in the sciatic region after extraction by the feet; Bruns considers that such paralysis in extraction by the feet must occur frequently, but possibly they subside very quickly.

(2) TRAUMATIC FACIAL PARALYSIS

The facial paralyses caused by birth trauma fall into two groups:—

(A) FACIAL PARALYSIS AFTER FORCEPS DELIVERY.

Paralysis takes place most frequently during birth by lesion of the peripheral facial trunk at its point of emergence from the



FIG. 35.—Facial paralysis of right side and contractions of the upper extremities resulting from subdural haematoma in the left frontal region. Forceps delivery.

stylomastoid foramen, or of its branches, either on account of direct pressure by the blade of the forceps, or indirectly, from a haematoma caused by the forceps and involving the nerve, by oedema forming in the region of pressure, and possibly also by simple stretching of the nerve (Falloux). The forceps may also result in a compression of the brain and a lesion of the facial area of the motor cortex. Usually these are cases of intracranial haematoma (fig. 35). Isolated

facial paralysis due to this cause occur but rarely. Usually in such cases a paralysis of the limbs of the same side is also present.

(B) FACIAL PARALYSIS AFTER SPONTANEOUS DELIVERY.

In spontaneous delivery facial paralysis can take place only when there is disproportion between the size of the fetal head and the maternal pelvis. In this way opportunity is given for the exercise of considerable pressure by the pelvic wall upon the skull. The paralysis therefore occurs chiefly in the various forms of flat pelvis, and in the position of the parietal bones characteristic of this form of pelvis, one being in front of the other (Ludwig, Kellner, Frank). The pressure is from the projecting promontory or from the symphysis, especially when exostoses are present behind the latter (Vogel and Gröte). Usually this occurs in cases of head presentation; only rarely of breech presentation (Vernier). In spontaneous delivery also the cerebral cortex in the region of the facial centre may be injured directly or by an extravasation of blood. In the great majority of cases, however, the injury affects the peripheral nerve and especially its extracranial portion, whether it be directly compressed by a bony process, or whether the shoulder be pressed against the point of exit of the nerve (Frank and Le Queux). Often in this region a tense swelling is found, recognizable as a hematoma in the upper attachment of the sternomastoid. In these cases a contracture of the sternomastoid in question may take place at the same time (Schultze and Stein). Also the oedema in the periphery of a point of pressure on the skull may affect the emerging nerve-trunk (Knapp).

It is possible that there are also facial paralyses dependent on lesions of the intracranial part of the nerve. In such cases haemorrhage at the base of the skull is suspected. This assumption is more than probable when the facial paralysis is accompanied by paralysis of the hypoglossal nerve (Schultze, Stein). The latter condition is shown by the fact that when the tongue is put out it points towards the affected side or lies in the mouth twisted towards this side. A simultaneous oculomotor paralysis may be recognized by ptosis (paralysis of the levator palpebrae superioris).

Traumatic facial paralysis is usually unilateral and only exceptionally bilateral (Seefigmüller, Edgeworth). Usually it is not a case of total paralysis but only of paresis as may generally be seen at once, owing to the crying of the child.

The paralysis does not cause the child any inconvenience, sucking is hardly ever affected by it. The prognosis, at any rate in uncomplicated cases, is very favourable. The time of healing is almost always much shorter than in birth injuries affecting the upper extremity. Often the facial paralysis disappears completely after a few days or is at any rate much improved. It seldom persists more than two to three weeks. But even when it has persisted four to six weeks complete recovery may still be expected. Briel saw a

case in which this did not take place for six months. Only very rarely do degenerative processes lead to the persistence of the paralysis (Parror and Treussier, Henschi, Stephan). Treatment is usually quite unnecessary. Should the paralysis not have disappeared after a fortnight electrical treatment is advisable.

The differential diagnosis between simple pressure paralysis and intracranial forms (cortical and basal) is usually facilitated by the complications which almost always accompany the latter, the general symptoms of intracranial haemorrhage, motor irritation and the simultaneous occurrence of other paralyses of the cerebral nerves and the extremities. Cortical facial paralysis is often not recognizable immediately after birth and increases in intensity during the first days. The usual distinction between peripheral and central facial paralysis by the involvement of the frontal branch of the facial nerve is usually difficult in the newborn.

If a facial paralysis be found after a perfectly normal birth, one suspects injury of the facial nerve which is occasionally incurred by adherent strands of amnion, i.e., an intrauterine origin of the paralysis (Geyl). A congenital facial paralysis may also be of nuclear origin. It then belongs to the group of cerebral nerve paralyses which are described as Nuclear Aplasia and Infantile Nuclear Dystrophy (*vide infra*).

(II) INJURIES OF BONES

(A) Injuries to Skull Bones

The elasticity of the flat skull bones of the newborn, and the ease with which they over-ride one another at the sutures, make lesions of the bones relatively infrequent, since, even when there is considerable disproportion between pelvis and head, the latter moulds itself successfully.

(1) IMPRESSIONS ON THE SKULL.

The impressions which are made during birth on the flexible and somewhat elastic skull-bones of the newborn child fall essentially into two types, which may be described as spoon-shaped, and grooved. The spoon-shaped impressions are flattened, not strikingly deep, and circumscribed; the grooved impressions have fairly steep sides, with the bottom projecting some way toward the inside of the skull (figs. 36 and 37). There are several varieties of each type, basin-like and cup-shaped, triangular, and crescentic impressions. The shape in each case depends upon the situation, on the shape of the bony process which has caused the impression, on complicating fissures and incomplete fractures.

The impressions are found most commonly on the parietal bone, and usually between the parietal eminence and the coronal or squamous sutures. More rarely, the parietal eminence itself is involved. The next most frequent position is the frontal bone, or both frontal and parietal bones. The temporal bone and the pos-

terior part of the parietal are but rarely affected. The diameter of the impression is usually about 3 to 4 centimetres. The skin over



FIG. 16.—Square-shaped impression. (After Baum.)



FIG. 17.—Grooved impression. (After Baum.)

the impression usually shows but little alteration, pressure marks and slight suggillations. Very frequently the impressions are combined with slight fracture of the bone, chiefly of the outer table, but sometimes of the inner table also.

The impressions are caused by disproportion between the head

and the pelvis, and result almost entirely from the prominence in narrow and flat pelvis. For this reason it is almost always the posteriorly situated parietal bone or the frontal bone which is affected. Much less frequently the pressure is due to the symphysis. Kuster has described a funnel-shaped impression caused by a coccyx which projected far into the pelvis. Usually the infants are born by breech-presentation. Impressions on the parietal bone are practically limited to cases wherein the head is born last. The frontal impressions occur relatively frequently in forceps cases but less frequently from direct pressure by the blade of the forceps as by the prominence on account of the occasional forcible extraction of the head through the pelvis. Similarly, the hand of the obstetrician, in pushing the head through the pelvis from the abdominal wall, may cause similar results.

The children usually suffer no ill effects from the impressions. No brain lesions generally take place. Intracranial hemorrhages, which are occasionally found associated with impressions, only occur quite exceptionally as direct results of the impressions, but are rather the results of simultaneous compressions of the skull and over-riding of the bones. Kuster observed fatal hemorrhage as a result of rupture of the middle meningeal vessels in a funnel-shaped depression of the temporal region. Also the frequently observed asphyxia must be considered to be less the direct result of the impression than caused by difficult delivery with contracted pelvis. The same applies to cerebral disturbances which sometimes appear later on.

Shallow spoon-like depressions may disappear spontaneously but with the funnel-shaped type complete restitution is exceptional. Slight levelling undoubtedly tends to take place during the first weeks of life, but a more or less obvious depression in the bone remains. On cosmetic grounds it is thus desirable to reduce the impression at least when it is situated on the frontal bone. Several methods are available.

Kerr recommends that the child's head be taken with both hands and considerable pressure brought to bear on the region opposite the depressed area; the latter then springs out sometimes, "just as a dent in a felt hat may be levelled out from within." Hoffmann recommends a similar procedure, "the skull bones are pressed near the edges of the depressed area, the two thumbs being on one side of the impression and the index and middle fingers on the other side. The fingers at first are at a considerable distance from the thumbs but are pressed approximately in the direction of the centre of the skull under the deepest part of the impression so that they try to meet the thumbs below the impression. The bone sometimes springs back to its normal position with a jerk; in other cases reduction at first takes place gradually." Hoffmann observed very good results from this method which sometimes only takes a few minutes but usually needs a few hours. The straightening of depressed bones with the air pump is but rarely successful.

Several authorities (Nicol, Newton) advise trephining and elevation of the depressed area of bone, always a somewhat radical measure. Boissard elevates the depressed area without trephining; he introduces a flat instrument (probe, spatula or something similar) at the suture, passes it between dura mater and bone and is thus able to reduce the depression from the inside. This procedure also should only be used in exceptional cases.

The most suitable operative method consists in elevating the depressed part from the outside. For this purpose Tapret, many years ago, used a corkscrew-like instrument which has more recently been re-introduced with modifications and improvements and its use has been attended with success (Vicarelli, Baumann, Schefzick, Häsch, Soli). The elevation may be achieved with a small cork-



FIG. 38.—Vicarelli's instrument for treatment of skull depressions. (After Soli.)

screw or a borer specially constructed for use in this operation, and fitted with a special arrangement to prevent the thread of the screw from penetrating too deeply (fig. 38). After disinfection of the skin a bistouri may be driven straight into the bone or this may be laid bare with a small incision, the borer is then applied. In shallow depressions the direction of the bore is towards the centre of the head, but with deeper ones the direction must be more oblique, in fact, almost tangential. Injury to the brain need not be feared. The bone is most easily raised when taken in hand immediately after birth; but the operation may also take place with good results twelve to twenty-four hours later; in one case of Vicarelli success was even attained after forty-five days. Out of twenty-three cases operated on by Soli using this method, three died and in these death did not result from the operation.

(2) FRACTURES OF THE SKULL.

(a) *Fractures of the Calvarium.*

The type of skull fracture relatively of most frequent occurrence, are the so-called fissures and cleavages of the parietal and frontal bones. They usually follow the direction of the trabeculae of ossification which run radially from the frontal and parietal eminences towards the periphery of the bone (Hartmann). Such fissures are frequently, but by no means always, associated with depressions and usually have a similar origin during birth through a narrow pelvis by pressure by the promontory or on account of injuries with the forceps or by the expression of the head when this is born last. The application of forceps very high up may lead to fractures of the orbital roof (Dittrich).

The fractures may be simple or multiple. They occur particularly in abnormally thin skulls and those in which ossification is imperfect, in which case the labour pains will alone be sufficient cause, even with small skulls. If the skull be hard then it is only liable to fracture when subject to the maximum of pressure; then the smaller the interstitial membranes the easier is the occurrence of fracture (Dittrich, Rosinski). Züsser describes a case in which after a perfectly normal birth (small head and wide pelvis) both parietal bones were fractured, obviously only as the result of the sudden and powerful driving of the head against the pelvic floor at the moment of the rupture of the membranes. Van Striethoven attributes one case of fissure of the parietal bone to the spasm of the os uteri after the administration of a large dose of ergot.

Fissures of the calvarium may not cause any symptoms at all. They are often not discovered until the post-mortem examination. Obvious dislocations only occur with very multiple and complicated fractures. In conjunction with fissures, internal and external haematomata and haemorrhages between dura and bone or under the periosteum, often occur. A rare but clinically important complication of fracture of the calvarium is the so-called spurious traumatic meningocele, the traumatic cephalic hydrocele. This arises when, in a fracture of the skull, there is not only tearing of the dura but injury of the inner meninges, and naturally also of the brain. These are usually cases of forceps delivery. The edges of the fracture of the skull bone are pushed away from one another by an intracranial extravasation of blood, which easily happens owing to the distensibility of the infantile skull. The cerebrospinal fluid gets under the scalp through this fissure, meninges and parts of the brain bulging out like a hernia.

The clinical picture of such a cerebral hernia consists of a soft tumour, usually in the parietal region, not usually becoming visible until a few days after birth; it then grows more or less quickly, and, in the course of a year, may grow as big as the child's head. Only a few such cases have been described up to the present (Billroth, Boyerthal, Rahm, H. Chiari, Elber and Schindler).

The prognosis is serious. Owing to operative interference and other injuries the danger of infection is considerable. Recovery, even spontaneous recovery, is quite possible; the cleft in the bone may close, and the protruding part of the brain undergo absorption, but in such cases as in true cerebral hernia there is the danger that porencephalic holes may be formed, which, according to the situation of the hernia, are followed by more or less severe symptoms of cerebral atrophy (paralysis, cerebral irritation, idiocy, &c.). The time for operation depends upon the symptoms present and on the progressive character of the condition. Puncture may, under certain circumstances, lead to permanent diminution or disappearance of the meningocele; usually, however, the results are only temporary. The best methods are the excision of the meningocele (Kronlein), and the radical operation of Kornig: Incision and closure of the bony fissure, either by raising the depression in the base or by freeing of the pericranium which is pulled over the fissure and sutured (Rahm).

(b) Fractures of the Base of the Skull.

Fracture of the base of the skull is an exceedingly rare birth-injury. The type which is of greatest relative frequency is due to separation of the condylar and supra-occipital parts of the occipital bone. The mechanism of this injury is such that the supra-occipital is torn away from the other parts, or is pushed against them so hard that it over-rides them internally. This happens as follows: The occiput is pulled downward while the head is unable to follow; at the same time there is a compression in the fronto-occipital direction which presses the supra-occipital under the condylar portion. When the head is born last, the mechanism is quite similar (Kustner). The injury occurs in forceps delivery or in extraction, but may also occur in spontaneous delivery with flat pelvis (Hartmann). The results of such a fracture are very serious, partly as a result of the accompanying hæmorrhage in the region of the medulla oblongata, partly on account of the narrowing of the foramen magnum which always takes place. The supra-occipital in being pressed forward, may even guillotine the medulla.

(c) Fractures of the Face Bones.

Fractures of the roof of the orbit, the upper margin of the orbit, and of the lachrymal bone, are observed in forceps delivery, fractures and dislocations of the mandible in extraction of the after-coming head (Abrahams).

Intra-natal injuries sustained by the mother or by a second person may closely resemble the birth-injuries to the calvarium occurring with contracted pelvis. In general, the injuries caused by a sudden external agency are more sharply circumscribed than birth-injuries; the fractures are usually multiple, the break is constantly lying more in the centre of the depression, but none of these are certain signs, from the forensic view, of external agency; the same

holds true for injuries to the skull after breech delivery. According to Fritsch, the characteristics of any skull injury arising from the child's falling on the floor are as follows: the fracture begins in the course of a suture, and proceeds from there, almost at right angles, to the middle of the bone. Usually only one fissure is caused—almost always in the parietal bone. According to Dietrich, this finding is not pathognomonic of precipitate delivery, its significance depending upon the course of labour and the overlying of the skull-bones.

(B) Injuries of the Skeleton of the Trunk

The vertebral column of the newborn is extraordinarily flexible and elastic, and as yet partially cartilaginous. For this reason it withstands very great bending and twisting without permanent injury (Gott). While within the vertebral canal, extradural, intermeningeal or intramedullary haemorrhages may occur throughout the whole spinal cord, the injuries affect the vertebral column in the cervical and upper thoracic region only. They occur almost exclusively with extractions in the course of delivery with an after-coming head, and only quite exceptionally in head presentations, when the delivery of the shoulders is hindered. The first and second cervical vertebrae usually escape (only O'Connor describes a case of fracture of the odontoid process), otherwise all the remaining cervical vertebrae may be injured. Lesions of the sixth cervical vertebra are the most frequent, since its body is the least protected; the disc between the sixth and seventh vertebra is almost twice as broad as that between the fifth and sixth. In pulling at the shoulders, the less secure connection of the lower and the upper epiphyses of the sixth cervical vertebra is very easily severed (Höflner and Czernowiz). Injuries of the cervical vertebral column consist, chiefly, according to Stolzberg, in the tearing off of the capsule of one of the lateral joints, also in rupture of neighbouring parts of the intraarticular ligament and rupture of the vertebral body through a plane of cartilage. Sometimes, however, the vertebrae and discs are not injured at all. The injury takes place owing to a pull which deviates from the longitudinal direction; its result is usually dangerous to life. Owing to the difficulty of diagnosis in such injuries, no one has ascertained whether life can be preserved. As a rule, the subsequent haemorrhage in the vertebral canal will be fatal. Birnbaum describes the symptoms thus: Breathing either fails to begin at all, although the heart continues to function for a time; or, breathing ceases immediately, before any disturbances of the heart's function appear. It may happen that haemorrhage is at first checked, but is started again by movement. Although life is usually very short, it may last for some days (in one case under my own observation, two days, and in one case of Ahlfeld's, so long as nine days).

Ribs fractures may occur during forcible version, as for instance in rough gripping of the thorax in extraction of the trunk (Winter). Stumpf describes a case where all the true ribs were fractured together with the right superior ramus of the pubis, after very difficult version from an oblique presentation. In general, fractures of the ribs are very rare birth-injuries.

(C) Injuries of the Upper Extremity

(1) FRACTURE OF THE CLAVICLE.

Fractures of the clavicle are among the relatively frequent birth-injuries. According to Muus, they occur in 1/3 per cent. of all births.

Fracture of the clavicle occurs in spontaneous delivery and about twice as often in the children of multiparae as in those of primiparae. This indicates that the fracture may be caused entirely by the power of the pains, as the shoulders are passing through the pelvis. An accessory factor is the manual pressure exerted by the midwife on the perineum during delivery of the shoulders. This has been suggested by Riether, and proved by Hauch from experimental researches on the dead subject. As a rule that clavicle is broken which presses against the symphysis during delivery. The mode of occurrence of the fracture is probably as follows: The anterior shoulder is pressed against the posterior, flat portion of the symphysis in such a way that the clavicle is bent beyond the limits of its elasticity, and breaks. After this, the shoulders are delivered, and the clavicle springs back to its normal position. This indirect mode of origin is certainly far more frequent than the direct, which may occur when the shoulders are pressed down in order to free the arm or in the delivery of the head. For this reason, fracture of the clavicle is much more frequent in head than in breech-presentations. The separation of the sternal epiphysis of the clavicle may occur when the arm is pulled forcibly.

The clinical symptoms are exceedingly indefinite. Only rarely is the characteristic sinking outwards and forwards of the shoulder present as in later life. Usually the position of the shoulders is perfectly normal. The infants move the affected arm almost exactly as they do the sound one, and only rarely show obvious pain when the position of the fracture is pressed upon, or when the arm is pulled about. Nevertheless, weakness and pain of the arm are sometimes seen. Possibly in such cases there is a simultaneous injury of the brachial plexus or shoulder joint. Large haematomata and marked deviation of the fragments are only seen in exceptional cases. Sometimes a slight kinking or break in the continuity of the bone may be felt, together with abnormal mobility. The most reliable symptom in recent cases is crepitation, which is felt when the finger is moved along the bone, especially when the arm is considerably abducted. Recent fracture of the clavicle is frequently not diagnosed until it is looked for; otherwise it is usually missed or not discovered until an easily palpable callus has been formed.

Fracture of the clavicle occurs chiefly in the middle third, or between the outer and middle thirds of the bone, sometimes also near the acromial end. It is usually a case of simple transverse or oblique subperiosteal fracture, or simply of greenstick fracture. On the sixth and tenth day, distinct callus formation has usually

superficial at the point of fracture. The small, hard tumour is often quite striking.

The prognosis of clavicular fracture is very favourable. It usually heals spontaneously without any disturbances of function. Even when, at first, there is a dislocation, this is no disadvantage, because, with development of the thorax, the clavicle tends to assume its original shape (Reither). The callus formation also tends to disappear entirely during the first months. In view of the



FIG. 36.—Splint for fracture of the clavicle. (After Spitzzy.)

favourable prognosis, treatment is really hardly necessary. The majority of clavicular fractures heal perfectly without any bandaging. In severe dislocations, Reither recommends that a jacket should be put on, sewn up at the back and sleeves. The upper arm is laid on the thorax, and the forearm bent at the elbow and fixed to the front of the thorax, so that the hand, with the fingers extended, lies on the sound shoulder. With severe dislocations, the arm may be fixed in this position by blue bandages, after a wedge-shaped pad has been put into the axilla. The bandage is kept on for a week or two. Spitzzy recommends a cross-shaped splint, the

vertical arm of which lies at the child's back, and to the horizontal arm of which both forearms are bent at right angles and fixed, the upper arm being adducted; the fragments are separated by forcible forward motion of the forearm (fig. 39).

(2) INJURIES TO THE SHOULDER GIRDLE.

Fractures and separations of the epiphysis are observed at the neck of the scapula as a result of forcible rotation of the arm, also transverse fracture through the scapula, and separation of the acromion. All of these are rare injuries.

Dislocations of the shoulder joint are also rare in the newborn, since, with all movements which might cause such a condition, separation of the epiphyses tends to occur. When the arm is pulled out, as is done in brutal attempts at delivery, dislocation never occurs, but always separation of the epiphysis (Kratter). The separation of the epiphyses in the newborn corresponds to dislocations in the adult (Küstner). A case of true subcoracoid dislocation is described by Olshausen. Of recent times, Fink and G. Eckstein have described intracapsular dislocation of the humerus as a birth-injury. In these cases, the humerus appears abducted and internally rotated, its head projecting somewhat forwards. The treatment of this injury consists in fixing the arm in abduction and external rotation, after correction of the pronation of the forearm and the palmar flexion of the hand and fingers. Eckstein's case was complicated by a birth paralysis due to injury of the brachial plexus, which healed completely with suitable treatment.

Possibly the above injuries permit of another interpretation. Lange describes the occurrence of dislocations of the shoulder, which occur in forcing of the back-stretch arm, and which may simulate a birth paralysis. The pathognomonic position for such a dislocation is as follows: The upper arm is pushed forwards, abducted and internally rotated. The first two changes in position are hidden by compensatory rotation of the scapula; the marked internal rotation is always obvious, however. "If, a few days after birth, a child whose hyper-extended shoulder had to be freed, does not seem to be able to move this arm as well as the other, the first thing to do is to ascertain whether the musculature functions or not. Should it appear that no injury to the brachial plexus has taken place, and that the region of the shoulder joint is tender on pressure, an X-ray picture should if possible be made. In this way, a dislocation or a fracture in the ossified part of the humerus, may be ascertained or excluded. Injuries to the cartilaginous part of the humerus cannot be proved by X-rays with the same certainty, and this is only possible when there is considerable displacement of the fragments. Should this not be the case, careful movement of the arm, consisting of internal and external rotation, should be carried out. Soft crepitation or abnormal mobility in rotation, indicates the separation of an epiphysis. Should this examination

also be negative, the case is then probably one of dislocation of the shoulder joint. Should it then be shown that external rotation and raising of the arm backwards is particularly painful, then the diagnosis is established."

The method of treatment recommended by Lange for dislocation of the shoulder is as follows: The arm is first bound to the thorax and the pain in the shoulder is relieved by frequent changes of dressings of lead lotion. In order to prevent a very serious shrinkage of the jointcapsule, the arm is abducted at right angles a week after birth, pulled back as far as possible, and at the same time externally rotated as much as possible. In this position, the arm must be fixed, twenty hours a day for four weeks, either with a sand-bag, or better, by being embedded in plaster-of-Paris or something similar. From the second week onwards, light massage and passive movement may be undertaken such as lifting anteriorly, laterally, and backwards, also internal and external rotation, with fixed shoulder-girdle.

(3) INJURIES OF THE UPPER ARM.

Fractures of the upper arm are relatively frequent birth-injuries. The fracture of the shaft of the humerus usually occurs at about the middle or upper-third of the bone, and is usually transverse. The dislocation tends to be slight. The fracture occurs owing to the method used to free the arm, particularly when the arm is pulled down with two fingers only, instead of four, or when the arm is pulled with hooked fingers (Smith). Fractures of the humerus may also occur in spontaneous delivery from head-presentation, by light pulling on the head, or by the manoeuvres customary in every delivery (Jäger).

Separation of the epiphysis at the upper end of the humerus takes place not less frequently, an injury which, as has already been said, is frequently mistaken for a dislocation. When the upper-third of the arm is subjected to a transversely directed force, then the junction of the epiphysis forms the point of least resistance and gives way. When, as is usually the case, the periosteum tears off then a piece of the upper end of the shaft projects through the tear. If the pressure causing the injury acts from above then an axillary or subcoracoid dislocation is simulated, if it acts from the axilla the condition resembles a retroglenoid dislocation, since the shaft projecting through the tear in the periosteum feels very much like the head of a dislocated humerus. Küster points out that in such cases the absence of flattening of the shoulder may be utilized in differential diagnosis, and that a constant sign peculiar to the newborn may be observed in the inwardly rotated position of the humerus. Besides this the previously mentioned dislocation of the shoulder must be considered while making the diagnosis.

Nerve lesions must be mentioned as important, although relatively rare complications. These occur most easily in separation of

the upper epiphysis but may also occur in more distally situated fractures. Individual nerves may be paralysed by the pressure of fragments or by masses of callus. Loviot describes a case of isolated paralysis of the musculo-cutaneous nerve and Dollinger and Fiath paralysis of the musculo-spiral nerve.

Fractures with slight displacement and without complications may heal spontaneously or with a simple bandage without after results. Even badly healed fractures must not be regarded too seriously since the deformity tends to disappear entirely in the course of growth. Fractures of bones in the newborn usually heal very quickly with considerable formation of callus (fig. 40). If with severe displacement bandaging is thought desirable, and really



FIG. 40.—Fracture of the upper arm with considerable formation of callus.

fills its purpose, it will be found that the setting of the broken arm on a longitudinal splint is not very satisfactory.

Dollinger recommends the use of a plaster-of-Paris splint: the upper arm is abducted to an angle of 130° , the forearm flexed at right angles and supinated. The splint reaches from the lower-third of the forearm right to the axilla, it is bound to the thorax here, and is kept close to it as far as the twelfth ribs. The arm portion reaches half round the limb, the body part reaches to the nipple in front, and to the angle of the ribs behind. Brewin has evolved a modification of Dollinger's splint, and also suggests that if treatment is to be limited to bandaging the extended limb should be bound to the body in the position of "attention" instead of the simple bandaging of the upper arm to the thorax. Herff and

Oeri have invented a suspension apparatus which may be applied easily by even unskilled persons and which does not interfere with sleep and feeding. Spitzzy recommends that both upper arms be elevated horizontally and fixed to a splint behind the back; the forearms are bent at right angles and fixed to the splints, which pass upwards from the ends of the main splint (fig. 41).

(4) INJURIES OF THE FOREARM.

Fractures of the forearm appear to occur far less frequently than those of the upper arm. They have recently been described for



FIG. 41.—Splint for fracture of the upper arm. (After Spitzzy.)

the first time by Pforte. In two cases observed by this writer both bones were broken near the middle. The injury may occur in freeing the arm after version and extraction when the force is applied right in the middle of the forearm; a supinated position of the arm may be a predisposing factor for this injury. The freeing of the arm does not need to be particularly difficult. Perhaps the injury is more frequent than has been supposed. Pforte has also observed fracture of the radius alone in a case of difficult freeing of the arm. The best treatment consists in setting the forearm in a supinated position.

Dislocations are rare. Von Winckel mentions a case of dislocation of the hand.

(D) Injuries of the Lower Extremity

(1) INJURIES OF THE THIGH.

Dislocation of the hip is one of the rarest birth-injuries. It can only occur when in breech presentation the leg is thrown upwards and a powerful downward force is brought to bear on the neck of the femur (Küstner).

Separation of the epiphysis at the upper end of the femur is also very rare and particularly difficult to recognize. Plörie observed one such case after version and extraction. The diagnosis could not be made until three weeks after birth when callus was detected by X-rays. When such injuries are not recognized they may subsequently simulate a congenital dislocation of the hip or lead to the appearance of *coxa vara*.

Relatively the most frequent injury of the lower extremity and one of the commonest of all birth-injuries is fracture of the femur in its upper half. It occurs in the artificial delivery of breech presentations, when the finger or instrument used for extraction does not lie exactly in the groin, but slips on to the thigh. In this fracture the displacement is usually considerable, the upper portion being abducted and the lower adducted. Although in this fracture, as in all fractures in the newborn, the tendency to heal is very strong and straightening of the displacement is anticipated, early correction of the latter is none the less much to be desired. That this is not easily obtained may be judged from the large number of methods of treatment given.

The oldest method, and one which has been recommended again recently, consists in flexing the leg at the hip-joint and binding it to the body, so that its anterior surface is separated from the body by a layer of cotton-wool and the foot lies over the shoulder of either side (Credé, Küstner, Dollinger, Helferich, Zancarini, and others). With severe displacement, however, such an arrangement is not very effective. Of the various splints which have been proposed the plaster-of-Paris dressing recommended by Dollinger deserves mention: the thigh is flexed at the hip to an angle of about 100°. The dressing consists of an anterior and a posterior splint which both reach from the foot to the umbilicus. The limb part of each splint half encircles the limb, the body part extends on the injured side forwards and backwards somewhat above the middle line; the dressing is changed daily. Isbister uses an aluminium splint. Riese recommends a traction bandage: the leg lies thrown upwards and internally rotated with the outer side forwards, the knee-joint being slightly flexed, it is extended by means of a strip of adhesive plaster attached to the sides of the leg; in the under part of this is an elastic band passing under the foot forming a sling over the nape of the infant's neck. Other simple traction

bandages have been proposed by Landau and Jones. The suspension apparatus devised by Oeri may also be used in fracture of the femur.



FIGS. 42 AND 43.—Splint of fracture of the thigh. (After Spitz.)

Spitz uses iron splints 2½ cm. wide, which are riveted together at one end with their wide surfaces approximated. The point of union is opposite the end of the sternum. The legs are separated equally from the middle line, and in this position the splints are fastened to the front of the legs; they follow the lines of the most comfortable position for the child; hip and knee flexed and a right-angled bend for the dorsum of the foot (hinge splint). The legs

are then bandaged to these splints. Owing to the reciprocal separation the splint holds very well, and with raised position of the legs soiling of the bandages is avoided (figs. 42 and 43).

Fractures of the femur may also be observed in spontaneous delivery. Henning and Green observed spontaneous fracture in every case of face presentation in which the thigh was made immovable by being wound round by the umbilical cord. Vanerek observed it in prolapse of the foot near the head, and Stumpf during the delivery of the body in a head presentation. Engel mentions a case of spontaneous fracture of both femora in a breech presentation.

(2) INJURIES OF THE LEG.

Fractures of tibia and fibula and dislocations of the knee-joint occur, as a rule, only in very faulty attempts at artificial delivery. Von Bunsen has described several such cases. These injuries may, however, occur in spontaneous delivery. Offergeld observed a dislocation of the knee-joint which obviously took place because during birth (breech presentation), not only flexion of the knee occurred, but also abduction at that point. After the spontaneous delivery of the breech the lower part of the leg was seen to be flexed at the knee and thrown upwards to the lateral side of the thigh. Hoffa mentions a case of fracture of the leg, a hand's breadth above the ankle-joint, probably occurring from the foot being caught against the pelvic wall and leading to the formation of a pseudoarthrosis. Hayashi and Matsuoka, who compiled thirty-one cases, consider that the cause of these fractures may be found in disturbances in the endochondral ossification which result in part of the diaphysis remaining cartilaginous. Intra-partum fractures of the leg are frequently combined with malformation of the lower limb (malformation of the fibula, absence of toes and metatarsal bones). Fractures occurring in this way and recognizable owing to shortening and angular deviation of the affected limb should receive surgical treatment as soon as possible. The fractured ends must be freshened, sutured, and set and the tendo Achillis lengthened to prevent pes equinus. Owing to the non-appearance of bony consolidation, however, the results of this operation are usually imperfect.

(III) INJURIES OF THE INTERNAL ORGANS

(A) Injuries to the contents of the Skull and Vertebral Canal (Injuries to the central Nervous System)

(1) INJURIES TO THE CONTENTS OF THE SKULL.

(a) Intracranial Hemorrhages.

The hemorrhages affecting the central nervous system, which are important in the pathology of the newborn, are almost entirely of traumatic origin, and will therefore be dealt with in connection

with the other birth-injuries. The rupture of vessels which give rise to hæmorrhage are, so far as they concern larger vessels, direct consequences of birth-injuries. Smaller extravasations of blood owe their origin chiefly to the congestion caused by labour, and are thus indirectly attributable to the action of forces during the birth process. Congestion, for its part, exercises a considerable influence on the intensity of the hæmorrhage caused by the direct trauma, so that a clinical differentiation between traumatic and congestive hæmorrhage is not possible.

Hæmorrhage between the Brain and the Roof of the Skull.

Hæmorrhages situated outside the brain but within the skull cavity are either epidural (extradural) between the skull-bones and the dura or subdural, between dura and arachnoid or subarachnoid in the reticulum of the pia mater, on the upper surface of the brain.

Hæmorrhages between bone and dura lead to the existence of the so-called internal cephalhæmatomata (see p. 182).

Hæmorrhages in the subarachnoid space (lepto-meningeal hæmorrhages) extend to the convexity of the cerebral hemispheres, and from the medial margin diminish in intensity at a lower level. The larger extravasations are usually bilateral, but often chiefly on one side, rarely entirely unilateral. Small lepto-meningeal ecchymoses are probably not rare in version and forceps delivery of the child. Sometimes they are accompanied by a subdural hæmorrhage. Ziehen points out that on account of the lepto-meningeal hæmorrhages occurring intra-partum being most frequently situated in the parietal regions, attacks of Jacksonian epilepsy may occur. In view of the frequent localization of the injury in the neighbourhood of the great longitudinal fissure it is particularly the leg centres which are affected, usually on both sides, but sometimes only on one. In the newborn child the symptoms of larger hæmorrhages are probably mixed, as a rule, with those of the accompanying confusion of the brain; they consist in symptoms of transitory motor irritation spasms, hypertonus, and disturbances in sensation. Smaller hæmorrhages must often take their course without causing any symptoms.

Among the intracranial hæmatomata of the newly born child, by far the most important are the subdural hæmorrhages (dural hæmorrhage and inter-meningeal hæmorrhage).

Since during later life such hæmorrhages only occur quite exceptionally, their mode of origin during birth must be quite characteristic. The aetiological factors which give rise to the peculiar qualities of intra-meningeal hæmorrhage are: the compression of the skull, which is considerable from disproportion between the size of the skull and the width of the pelvis, in narrow pelves, with the rigid soft parts of primiparae, particularly when forceps are applied, and also the overriding of the skull bones during delivery (Kudrat).

The injuries to the vessels which lead to hæmorrhage—that is,

the sources of the blood—are not always the same. In some of the cases the hemorrhage is derived from the veins opening into the longitudinal sinus, which may be injured in sudden overriding of the bones. Kundrat has pointed out that a hemorrhage only occurs when the skull bones are thick and hard as far as their edges



FIG. 48.—Flora of the skull. *x*, larger and smaller tears in the tentorium cerebelli; *y*, injury of *V. mening. med.*; *y*, sagittal in the tentorium cerebelli. (After Boverius.)

and when the interstitial membrane is not too narrow. The rigid bones form the points on which the pressure works and override one another, the superior longitudinal sinus is compressed, the veins opening into it are stretched, bent, and finally torn. Ruptures of the sinus itself are rare. When the margins of the skull bones are soft, and with wider interstitial membranes, displacement does not take place, because the pressure of delivery compresses the brain together with the skull. If the bones be rigid and the interstitial

mimbranes very narrow, no displacement of the skull bones, or only a very slight one, can take place. In a way similar to that in which the parietal bones override one another, the upper part of the occipital bone may be pushed under the parietal bones so that the transverse sinus situated in the lateral part of the lambdoid suture may be stretched and the veins opening into it torn. Besides the injuries of the venous channels caused by pulling and tearing the obstruction of the circulation due to compression of the skull may cause ruptures. In haemorrhage into the lateral ventricles which occur from the straight sinus it is probable that congestion is the only factor concerned (Seitz).

The rupture of the veins in the neighbourhood of the sinus is not the only cause of the haemorrhage. A considerable number of subdural haemorrhages depends, as has been shown by the researches of Beneke, on tears of the tentorium cerebelli. By means of a suitable dissection tears of the tentorium may be demonstrated in a large number of dead infants (average 17 per cent.) (fig. 44). Potz distinguishes three forms of these injuries. In the most extensive form a transverse tear is found if the free edge is situated near its middle point. The haemorrhage arises from the veins, which are present in large number in the edge of the tentorium. In the intermediate type a surface tear is found on the upper layer of the tentorium. In the mildest form the haemorrhage occurs between the layers of the tentorium; it has no clinical importance, as in this case there is no haemorrhage into the free skull cavity. Tears of the tentorium may also be combined with injuries to the sinus (Blaereisen). The cause of the tears of the tentorium is the lateral compression of the infant's head by which a stretching of the falx is produced. Usually the anatomical healing of the tear in the tentorium takes place without any difficulty, and apparently so smoothly, that after two or three weeks hardly any trace of a scar remains. Only when congestion occurs, after more serious injury and prolonged delivery, do large quantities of blood reach the skull cavity. Besides the situation and degree of the injury there is added yet another aetiological factor, congestion, which is not only of importance as regards the occurrence of haemorrhage, but also affects the degree and duration of that condition, whether the lesion be in the sinus or in the tentorium. The asphyxia, which occurs after birth partly as a result of the protracted course of delivery, and partly owing to the rise in intracranial pressure, also tends to cause a more lasting haemorrhage, and may thus be both cause and effect of the extravasation of blood.

Subdural haemorrhages, at least those which give rise to cerebral symptoms, occur more frequently after difficult labours, and especially after forceps delivery. Out of thirteen cases of Seitz ten were forceps deliveries; half of the cases were in elderly primiparae, and in a quarter of the cases the pelvis was contracted. Also in the cases of Beneke and Potz the number of patients in whom delivery was effected by means of forceps was considerable (60 per

cent.). The eleven cases of Bauereisen were all those of forceps delivery. It is noteworthy that nine of these were cases of extraction from breech presentation, after which Fritsch had noticed dural hæmorrhages. Bauereisen is of opinion that the Vest-Smellie manoeuvre more easily leads to intracranial injuries than an extraction with forceps; for, whereas the pressure exercised on the skull in the latter case can be graded, the attempts at extraction of the after-coming head often takes place most precipitately. Although subdural hæmorrhages may occur after operative delivery in which the technique was perfect, the skill of the operator must also be an important factor.

Subdural hæmorrhages also occur, however, after easy spontaneous labours of short duration. Injuries of the tentorium and of the sinus are even more frequent in such cases. It sometimes appears that the power of one expulsive labour pain is sufficient to cause an injury (Seitz, Beneke). Cases of fatal hæmorrhage have even been observed in spontaneous and quick deliveries and wide genital tract of multiparæ. The lateral compression, so important a factor in the occurrence of tears of the tentorium, may be due to unsuitable support of the perineum (Stöckel). A certain importance is also attached to the quality of the walls of the vessels which in premature infants may be injured by comparatively slight trauma.

Finally, isolated cases of intracranial hæmorrhages of intra-uterine origin have been observed by Osler in a child born in the seventh month of pregnancy that was delivered after its mother had succumbed to typhoid without there being any pressure on the head; by Seitz in a macerated, spontaneously delivered, mature foetus; by Kusner in a child delivered by Cæsarean section before the commencement of labour. Bauereisen believes that in such cases intra-uterine injury is not necessarily present, since the hæmorrhage may be caused by compression of the skull by the gap in the uterus.

The extravasations of blood, resulting from the injuries named above, have very different symptoms according to their situation. Seitz, to whom we are indebted for a detailed description of the symptomatology, distinguishes the four following types according to the situation of the hæmorrhage:—

(1) *Supratentorial Hæmorrhage*.—This is derived from injury of the superior longitudinal sinus, transverse tears of the free edge of the tentorium, or from injuries of its upper layer. The blood flows over the cerebellum around the cerebral peduncles and the medulla is like a skin; in other cases it prefers to flow into the temporal fossa or around the occipital lobe. Supratentorial hæmorrhage is usually unilateral.

(2) *Infratentorial Hæmorrhage*.—The blood flows from the transverse sinus or from the margin of the tentorium, and flows over the cerebellum around the cerebral peduncles and the medulla oblongata, and even into the spinal canal.

(3) *Mixed Types of Supra- and Infratentorial Hæmorrhage*.

(4) *Hæmorrhages into the lateral ventricles*, in which the blood

comes from the region of the straight sinus (*sinus tentorius*), of the inferior longitudinal sinus, and of the veins of Galen.

In a number of cases death occurred a short time after birth, either when still in an asphyxiated state or when the child has been caused to breathe by attempts at resuscitation after the first few breaths. These are mostly cases of combinations of intermeningeal hæmorrhage with cerebral hæmorrhage or contusions, or of serious injury to the medulla oblongata. With larger intracranial extravasations of blood, such as are observed after extraction from breech presentation, death usually takes place at once.

Frequently, however, the infants rally immediately after birth and the symptoms only come on gradually. In such cases it must be assumed that the hæmorrhage from the ruptured or torn vessels, which is at first slight, persists after birth and even increases. This may easily occur after the removal of the pressure of birth, whether it be that the increase in hæmorrhage results from the persistence of venous congestion or from stimulation of the vasomotor centre leading to a rise in the blood pressure. The increasing flow of blood leads to an increase in the intracranial pressure. At first a certain compensation occurs, the sutures and fontanelles being widened and pressed outwards. Soon, however, the tension reaches such a degree that the limited resistance of the skull exceeds that of the brain. Symptoms of cerebral pressure then occur. Sometimes the infants are very restless during the first day, they cry much and painfully, and will not drink. Obviously stretching of the very sensitive dura gives rise to pain. Should the raised intracranial pressure act on the medulla oblongata injury of the respiratory centre may inhibit the child's crying. Also where there is confusion of the brain and disturbances of sensibility the child will behave quietly from the beginning. The slowing of the pulse, which in later life may usually be observed as a result of vagus stimulation, shows a tendency in the newborn to be absent, probably due to the low excitability of this centre. On the other hand, stimulation of the vasomotor centre is usually shown by the rise in blood pressure, which may be recognized by the strong full pulse and the accentuation of the second aortic sound. Spasm of the vaso-constrictors may cause the skin to become very pale, which may be followed by a transitory reddening during a convulsion. Dermographism may sometimes be noticed. The respiration is usually markedly influenced. It is usually somewhat deeper and longer, sometimes intermittent, spasmodic and accompanied by cries, sometimes, however, it is quiet and superficial.

At the autopsy the heart and great veins are found full of dark fluid blood. In the lungs widespread atelectasis is often found owing to the injury to the respiratory centre. Kundrat is of opinion that the condition of focal atelectasis in otherwise strong infants is extremely characteristic of intracranial hæmatoma; this indicates in the first place a hæmorrhage in the posterior fossa of the skull.

The symptoms which are clinically the most conspicuous are

the disturbances in the sensation and the signs of motor irritation. It is just these observations on children with intrameningeal hemorrhages which teach us that the newborn child is not merely a "subcortical reflex being," and that important functions are attached to the cortex. The restlessness and signs of pain of the initial stage gradually cease; the child falls into a somnolent condition, which may eventually change to a profound coma. It no longer reacts to external stimuli or cries when being changed or under similar circumstances; the need of food seems to disappear gradually. Even strong infants do not suck at the breast, although at first the introduction of a rubber teat on a bottle will produce sucking.

The most characteristic and the most important symptom for diagnosis are convulsions. Intrameningeal hemorrhages are certainly the commonest cause of convulsions during the newborn period. These consist of clonic spasms which occur at longer or shorter intervals, and are frequently started by external stimuli; they may affect the whole musculature of the body. Not infrequently the muscles of respiration are also involved. All transitional types occur, from almost unnoticeable twitches, in which the appearance of the child hardly alters, to severe epileptiform fits with tonic rigidity of the muscles of the body and marked cyanosis. The reflexes are usually much increased and in fact the tendon reflexes are involved as well as the skin reflexes. Frequently there is marked hyperonus, often greatly increased mechanical excitability of the muscles of the face (Chvostek's sign) and of the limbs. The spasms are usually bilateral, but they may be limited to one half of the body or to a definite muscle group according to the situation of the hæmatoma. In this way the symptoms of motor irritation assume the character of localized symptoms. Thus in the region of the facial nerve of the side opposite to the hæmatoma twitches often occur, to which symptoms of paralysis are soon added. In the contralateral arm and leg, and also in the sternomastoid, spasms and convulsions of a hemiplegic type occur. Sometimes the upper extremity is at first more affected than the lower. Trismus is not very frequently observed. In the eye, ptosis, strabismus, nystagmus and miosis occur, which in fatal cases tend to change to mydriasis and anisokoria. Miosis and ptosis are usually found on the same side as the hæmorrhage, but do not permit of any definite conclusion concerning the localization of the latter. The hemiplegic type of spasm and paresis of the limbs is also obliterated since similar symptoms occur on the opposite side of the body, probably as a result of compression, congestion and œdema of the other hemisphere. Yawning must also be looked on as a cerebral symptom, and occurs in such cases with striking frequency.

The period of irritation is finally succeeded by a period of paralysis. The muscles affected show marked paresis, and the tendon and skin reflexes disappear. In the course described death usually takes place between the fifth and eighth day. It is usually

death from failure of the respiration in a condition of coma. Frequently death is due to pneumonia caused by aspiration, a complication which, owing to the somnolent condition of the child, can hardly be avoided, especially when vomiting occurs.

The issue is, however, not invariably fatal. In some cases a slow improvement begins during the period of irritation. The convulsions become less frequent and finally cease completely about the end of the first week. The child begins once more to react to external stimuli and to take food. The improvement usually occurs on the third or fourth day. Should the symptoms still increase after this time a fatal termination must be expected.

The picture described, which is subject to many variations, corresponds essentially to a supratentorial haemorrhage. Infratentorial haemorrhage usually leads to death with symptoms of asphyxia either immediately or a short time after birth. Even when the children do not die at once they rarely survive more than twenty-four to forty-eight hours. Seitz's case, which lived for six days, must be classed among rare exceptions. Even with haemorrhages into the posterior fossa some hours may pass at times before any dangerous symptoms occur. If the flow of blood be small at first, asphyxia after birth may be absent or only slight. The children sometimes still cry loudly. The restless condition and the cries of pain which precede the stage of motor irritation in cases of supratentorial haemorrhage are usually absent in these cases. Even the tension of the fontanelle is absent at first; this only then occurs when obstruction of the circulation and oedema take place secondarily in the anterior fossae of the skull. The convulsions may then start, their most common type being bilateral. Sometimes irritation of the respiratory centre is the most prominent symptom; respiratory spasms then take place with marked cyanosis. As a result of the haemorrhage spreading downward spinal symptoms develop: rigidity of the neck, opisthotonos, erection of the penis, and spasms of the limbs.

Haemorrhage in both fossae of the skull almost always leads to death on the first day; life is rarely preserved for a few more days. Haemorrhage into the lateral ventricles produces symptoms very similar to those of infratentorial haemorrhage. At first a rise in pressure in the cerebrum takes place. Porak and Katz observed spasms confined to the face in a case of haemorrhage into the right lateral ventricle. As soon as the blood has broken through into the fourth ventricle and into the spinal canal medullary and spinal symptoms preponderate.

The diagnosis of subdural haemorrhage is easy to form when the symptom complex is marked and by a consideration of the history. It must, however, be pointed out that simple contusio cerebri (see p. 216) may cause very similar symptoms, and that cerebral symptoms also occur in cases of hydrocephalus and meningitis. The protrusion of the fontanelles and sutures may naturally also occur in both these diseases. On account of the usual unilateral

situation of the hæmorrhage the skull is often asymmetrical, the half affected by the hæmatoma being swollen, and the sutures on this side gape more widely, though unilateral hydrocephalus or pyrocephalus may also occur. In such cases lumbar puncture may sometimes enable a diagnosis to be made. The cerebrospinal fluid may contain blood or may be coloured reddish, or, more frequently, brownish red. It must not be forgotten that the presence of blood in the fluid may be due to the injury of a small vessel during the puncture. Usually this fresh blood coagulates quickly, while that which has been added to the cerebrospinal fluid in the body remains for a considerable time uncoagulated. When hæmorrhage has taken place in the anterior fossa of the skull the fluid obtained by lumbar puncture may be perfectly clear and colourless; a normal condition of the cerebrospinal fluid, therefore, by no means contra-indicates the diagnosis of intermeningeal hæmorrhage. Diagnostic errors are sometimes unavoidable. In particular it is hardly possible to distinguish between a subdural extravasation of blood over one hemisphere from a hæmorrhage within the same. The assumption of a dural hæmorrhage is only justified by the much greater frequency of this condition. Smaller extravasations must frequently escape detection. They either produce no symptoms or their symptoms are lost in those of the condition of asphyxia.

Hæmorrhage in the Brain Substance.

Intracerebral hæmorrhages are much rarer than intrameningeal. No exact observations concerning their symptoms are available. They must closely resemble supratentorial dural hæmorrhage. According to the post-mortem observations of Couvelaire, cerebral hæmorrhages are found most frequently in premature infants, in whom the brain is much more frequently the seat of hæmorrhages than the spinal cord; in five cases (out of fifty-one autopsies) the hæmorrhage was twice in the cortex and three times in the central white substance and the basal ganglia; the infants had reached the age of from 2 to 18 days. The immediate cause of the hæmorrhage might be the congestion caused by asphyxia, which with the delicate vessels of premature infants might easily lead to rupture. In seventeen autopsies of newborn infants who died after forceps delivery, Couvelaire found a hæmorrhage in seven; curiously enough, this did not usually correspond to the point of application of the forceps, but was in the cervical cord or in the medulla oblongata. Larger hæmorrhages in the substance of the brain may also be combined with intrameningeal hæmorrhage.

Intracerebral hæmorrhage may occur already in the fœtus ante partum, in fact, through trauma which affect the pregnant uterus. Seitz describes a case of extensive congenital defect of the brain after hæmorrhage into the medullary substance of the cerebrum, probably the result of an injury suffered by the mother in the fourth month of pregnancy.

(b) Contusio Cerebri.

Symptoms of cerebral compression and motor irritation (tonic and clonic convulsions) occur occasionally after difficult labour (e.g., forceps delivery followed by severe asphyxia) resembling the symptoms of hæmorrhage, but the autopsy only reveals hyperæmia and cedema of the brain and meninges, such as is seen in infants who slowly succumb to symptoms of asphyxia. In explanation of these appearances Seitz suspects changes, such as those occurring in concussion of the brain, and suggests that in these cases injury of the cerebral substance is present, and which may be described as contusion.

It is highly probable that continued vascular congestion alone may be followed by serious lesions of the brain. It may sometimes be observed that infants, who remained in a condition of asphyxia for some time after birth, show during the first days of life a marked apathy, somnolence and desire for sleep (with frequent yawning), and also an insufficient demand for food. Symptoms of motor irritation may be entirely absent. The condition gradually improves, inasmuch as the infants become somewhat less lethargic and drink better. It is difficult to decide in individual cases whether the cerebral symptoms following asphyxia are due to cedema, to hyperæmia, or to minute lesions of the brain substance from vascular congestion, from contusion or from hæmorrhage.

Prognosis and Sequela of Intracranial Injuries.

As follows from the above the prognosis of fresh hæmorrhage in the skull cavity is a serious one, when it is of such extent and situation as to produce symptoms of disease within the first days. Some of the children, however, survive the immediate results of the extravasation; this is then absorbed and the life is saved. In other cases the hæmorrhage may be so slight and its situation so favourable that in the newborn child obvious and characteristic symptoms are slight, if not altogether absent. The important question then arises: will these birth-injuries which have either healed or occurred without causing symptoms have any permanent effects in later life?

Intracranial hæmorrhages are sometimes responsible for the occurrence of hydrocephalus. Through extensive extravasation of blood a condition may occur in which the draining off of the cerebro-spinal fluid is interfered with. To this an additional factor may be added, that the resistance of parts of the walls of the ventricles may suffer through extensive destruction of these parts (Engel, Fischer).

Certain cases of potencephaly may also be attributed to intracranial hæmorrhage during birth. In this connection hæmorrhages occurring ante partum as a result of injury of the mother during pregnancy are also of importance (Zappert, Seitz).

Intracranial hæmorrhage in the newborn may also be of great importance in the ætiology of nervous diseases which appear later. On account of lesions of the infantile brain destructive processes

may take place in the cortex. A connection is thus made with epilepsy and idiocy. Intracranial hæmorrhages, particularly intermeningeal, are of especial importance in the ætiology of infantile cerebral paralysis—even if their significance may be overestimated. It is by no means impossible that even contusions of the brain, and also the lesions of the cortex caused by asphyxia, may be of ætiological importance in this class of disease.

According to Zappert intermeningeal hæmorrhage is of importance in the ætiology of the following conditions:—

- (1) General rigidity with little or no dementia, usually without convulsions (Little's disease).
- (2) Paraplegic rigidity without dementia or convulsions.
- (3) Simple hemiplegia with mental deficiency and convulsions.
- (4) Bilateral hemiplegia with mental deficiency, spasms and, finally, pseudo-bulbar paralysis.

Of the above types (3) and (4) must be looked on as the typical results of intermeningeal subdural hæmatoma. The paraplegic types, the typical Little's disease, indicate a bilateral lesion. Perhaps it is just the subarachnoid hæmorrhages which, without obvious symptoms in the newborn, cause severe injuries of the motor and psychical cortical centres. Perhaps hæmorrhages in the cortex itself or mere contusions with mild hæmorrhage may also be concerned in causing these types of disease. In such lesions the pyramidal tracts suffer most, the fibres of which are still unmyelinated, and are changed to a condition of degeneration and arrested development. Perhaps this partially explains the fact that Little's disease is so frequently found in the premature child which is in an early state of development at the time of the birth-injury (Oppenheim). (*Vide infra*.) Undoubtedly a large number of cases, which we look on as congenital types of infantile cerebral paralysis, remain entirely latent during the first period of life, or only present the appearance of a more or less severe asphyxia. According to Ziehen, it is especially in cases of infantile paralysis following asphyxia that serious mental deficiency is to be feared.

Treatment of Intracranial Injuries.

Since intracranial extravasations of blood may heal spontaneously, treatment must be expectant and symptomatic. This applies equally to hæmorrhage, to contusion, and to other forms of cerebral injury. All external stimulation, both mechanical and thermal, must be avoided. The child should be left alone so far as possible and supplied with adequate warmth. Cold compresses (cooling apparatus, ice bag) may be put on the head. To counteract excessive motor excitability and possible convulsions suitable narcotics are given, chiefly chloral, as an enema (0·25–0·5), bromides internally (0·5–1·0 gm. a day), or calcium (calcium lactate two-hourly one teaspoonful of a 1 per cent. solution). Grunfelder

recommends (in utero) a combination of the two drugs (calcium bromide 20·0, aq. des. ad. 300·0; three times a day 10 gm. in milk). The subcutaneous injection of calcium is not advisable on account of the likely occurrence of necrosis; Müller and Saxl recommend calcium gelatine (Merek's "kalgine"); this is a safe preparation for the purpose if the ampoule be placed in boiling water for ten minutes before the injection.

Feeding necessitates great care, since in the stupefied condition of the infants aspiration and all its results may easily take place. With children who do not swallow well, milk is best administered by means of an oesophageal tube, and fluid by the rectum or by subcutaneous infusion.

With marked symptoms of cerebral compression lumbar puncture may be not only of diagnostic but also of therapeutic value.

More radical methods are only justifiable in subdural hæmorrhage in the anterior fossa of the skull. In such cases operative interference has been adopted, especially by American authorities, and certainly the results have several times been very successful. Thorough removal of the coagulum is only possible by trephining. Cushing proceeds as follows: the parietal bone is pushed back, and after freeing it from the bone the dura incised, the blood clot removed, and irrigation is carried out with saline. Simmons proposes a simpler procedure. He incises in the region of the coronal suture and avoids trephining. Seitz carries out the operation in such a way that after pushing back the scalp an incision 2 to 3 cm. in length is made parallel to the middle line and about 1 cm. from it. It is, of course, desirable that the operation should take place at as early a stage in the disease as possible, before symptoms of paralysis appear, or as soon as the progressive character of the symptoms of cerebral pressure has been discovered.

Should the child be born in an asphyxiated condition, this must be treated immediately so as to prevent so far as possible the dangerous venous congestion which results in the continuance of a hæmorrhage. Great care must, however, be taken in attempts at resuscitation. Schultze's swinging method is definitely contraindicated in such cases and must be replaced by gentler methods.

(2) INJURIES WITHIN THE SPINAL CANAL.

Birth trauma is the most important ætiological factor for hæmorrhage in the spinal canal just as it is for cerebral hæmorrhage. The hæmorrhage is situated either outside the cord, in which case it is chiefly on the ventral side of the lumbar cord (the blood may have flowed down from the cranial cavity or from the upper cervical segments), or it may be situated in the substance of the cord, in which case it is usually in the junction between the anterior and posterior horns (Schäffer, Zappert).

No great clinical significance is attached to smaller hæmorrhages, which are found so often in such abundance in premature infants.

After more considerable hæmorrhages cystic cavities may be left which may possibly have some connection with a subsequent syringomyelia (Schultze, Zappert, Birnbaum, Stolzenberg).

Of greater interest in the pathology of the newborn are those extravasations which are followed soon after birth by obvious symptoms of paralysis. The hæmorrhages within the vertebral canal and in the cord itself (hæmatomyelia) occur practically entirely after forcible extraction from a breech presentation. They may or may not be combined with injuries of the vertebral column.

Ruptures in the region of the vertebral column, which occur chiefly in the cervical region, almost always cause death a short time after birth, as they lead to widespread extravasations of blood into the vertebral canal which compress not only the cervical portion of the cord, but may also reach the medulla oblongata and the posterior fossa of the skull (Ruge, Hofbauer, Stolzenberg, Czyzewitz) (see also p. 168). Whether small extravasations of this origin may be absorbed spontaneously is a difficult point to decide. Sometimes the hæmorrhage seems to stop, but starts again owing to movements made by the child. Pasoot describes a case of rupture of the cord between the sixth and seventh cervical vertebrae; the child lived for six days and showed greatly increased reflex excitability of the legs (Bruns).

Extraction of the child may, however, cause rupture of vessels, extravasation of blood in the cord and its membranes without there being any injury of the vertebral column. Thanks to the flexibility and elasticity of the vertebral column of the newborn fractures but rarely occur, at least in its middle and lower thirds, when the delicate cord within it is damaged (Gött). Either widespread hæmorrhage follows and destruction of the cord itself, or the hæmorrhage is chiefly outside the cord between vertebral canal and dura, or in the arachnoid or subarachnoid spaces. If the hæmorrhage does not lead to immediate death a very typical picture develops (Mauthner, Litzmann, Gött, Lavatschek). The most striking symptom is a flaccid paraplegia of the lower extremities, which may also show a considerable swelling during the first days. Less frequently the upper extremities are also affected (Parrot). Often paralysis of the bladder and rectum occurs, and later atrophy and the reaction of degeneration take place and widespread loss of sensation. The prognosis is very serious, but not absolutely hopeless. It depends entirely on the situation and size of the portion of cord destroyed. The higher the portion affected by the hæmorrhage the less favourable is the prognosis. When the bladder is paralysed the child may die from the results of cystitis. If only the legs are paralysed, it is usually only a case of extra-spinal hæmorrhage with compression of the roots of the nerves. In such cases an improvement during the first year is to be expected. The ordinary treatment for paralysis may then be employed (baths, massage, electrical treatment, &c.).

(B) Injuries of the Eyes

Birth injuries of the eyes occur but rarely. According to the statistics of Wolff, only six cases of eye injury were found out of 39,000 births in the maternity clinic at the Berlin Charité Hospital.

Eye injuries are found almost exclusively after births from head presentation. Out of 112 cases of Wolff's only three were births from breech presentation. The great majority of cases are forceps deliveries and owing to a narrow pelvis. The injuries take place most easily when forceps are applied to the head, which is still high up, and occur either directly from the blade of the forceps or as a result of severe compression of the skull while the head is being pulled through the pelvis. The protected position of the eye in the orbit makes injuries infrequent even under these conditions.

It appears that in spontaneous delivery injuries may result from abnormally powerful pains causing great compression of the skull. In fractures of the skull bones and intracranial hæmorrhages, either in cases of forceps or natural delivery, the eyes may be affected sympathetically. Injuries of the eyeball hardly ever occur in spontaneous delivery. Ante-partum injuries may occur in the vaginal examination of the pregnant woman, especially when the eye is mistaken for the breech (de Wecker, Bock).

Impressions and fractures of the walls of the orbit may not cause any symptoms whatever. They are usually situated in the region of the frontal bone, in the roof of the orbit, and are usually unilateral. Fractures of the lacrimal bone, also of the inner wall of the orbit, and the root of the nose have also been observed. Sometimes skin wounds are found at the same time. Hæmorrhages in the orbit may cause exophthalmos, injuries to the ball, and paralysis of the eye muscles. The latter are caused by injuries of the muscles themselves or of their nerves. After difficult forceps delivery paralysis of the levator palpebræ superioris, the superior rectus and of the external rectus have been observed. In the cases described by Berger and Bloch scars on the lids and canthus, due to the blades of the forceps, have been seen associated with paralysis of the eye muscles. Berger is of opinion that the injury occurs owing to the blade of the forceps acting as a lever, in which process the orbit forms a fulcrum, and thereby causes rupture of the muscles in question, thus giving rise to dislocation of the eyeball. The differential diagnosis between nerve lesions of intracranial origin and muscular paralysis cannot always be made with certainty. Barneff observed bilateral ptosis after an easy spontaneous delivery. After a forceps delivery Reese saw symptoms of injury of the sympathetic; narrowing of the palpebral fissure, exophthalmos and miosis. In addition to these injuries infection may result in the formation of an abscess and the occurrence of phlegmon of the orbit.

Edema and swelling of the eyelids and their connective tissue are frequent, especially as part of the caput succedaneum after a face presentation. After forceps delivery tearings of the eyelids have been observed which heal with the formation of scars and

may even lead to the formation of ectropion. Emphysema of the skin may result from injury of the root of the nose. Goring mentions such a case which after puncturing healed under a dressing. Peters describes cases of rupture of the inferior lachrymal canaliculus.

Hæmorrhages in the ocular conjunctiva, which are very frequent, are always to be looked on as the ordinary type of hæmorrhage due to vascular congestion.

Wagenmann mentions eighteen cases of dislocation and avulsion of the eyeball. In a frequently cited case of Hofmann after spontaneous delivery and administration of ergot one eye was found to be squeezed right out of the orbit and hanging by one muscle alone. Bagge and Fage also observed displacement of the eyeball after spontaneous delivery. Quite similar cases have been recently described by Snell, Wicherikiewicz, Thomson and Buchanan, but none of these were spontaneous deliveries. The optic nerve is usually torn at some distance from the eyeball, the muscles either quite close to it or at a more or less greater distance. The prognosis is naturally bad; only Beaumont has seen recovery of sight after replacement of the eye during narcosis. Total enucleation of the eyeball has also occurred during birth, it being found afterwards on the floor or in the bedclothes. Exophthalmos may occur owing to narrowing of the orbit caused by fractures of bones or by hæmorrhage in the orbit (Wolff).

Opacity of the cornea occurs almost exclusively after forceps delivery and only rarely after difficult natural delivery. It is usually unilateral and rarely bilateral (Sidler-Huguenin). It is usually a case of diffuse bluish-white opacity of variable extent and degree, and is due to œdema or sometimes to slight infiltration of the cornea. It is not yet decided whether these alterations are to be considered direct consequences of the trauma, or results of increased intraocular pressure which may occur in protracted delivery from disturbances in the circulation within the skull cavity. The prognosis of diffuse opacities is favourable; they tend to disappear completely in course of time. Less favourable is the prognosis in another type of corneal opacity, the striped or ribbon formed, deep opacity, which occurs from tears in Descemet's membrane and in the posterior layers of the cornea (Ruggeri and others). These injuries usually lead to permanent opacity of the cornea, corneal astigmatism or keratoconus.

Backward displacement and cataract of the lens have been described as birth-injuries. The iris and ciliary body may be the seat of hæmorrhage. Iridotodolysis was observed by Blyss in a case of forceps delivery. Also congenital atrophy of the optic nerve may sometimes be traced to birth-injuries (Schmidt-Rimpler). Lesions of the optic nerve occur, when they are not of intra-cranial origin, from stretching and rupture of the nerve in the orbital cavity, even though, as in the majority of cases, the lesion is only partial (Koppen, Fisher, Sidler-Huguenin). Choked disc has

been demonstrated anatomically by Naumoff. Possibly it may also be the cause of subsequent atrophy of the optic nerve. At present no researches have been made concerning the occurrence of shrunken disc due to increased intra-cranial pressure from hemorrhages in the cranial cavity. Tears in the choroid have also been attributed to birth-injuries.

The frequent retinal hemorrhages must be regarded as results of obstructed circulation (*vide infra*). Besides these, intra-ocular hemorrhages may be found in the anterior chamber, in the vitreous body, in and under the choroid and retina and sometimes in the optic nerve; total hamophthalmus also occurs (Wintersteiner, Lange and others). Separation of the retina is also described after spontaneous delivery and is caused by extravasation of blood. The prognosis of intra-ocular hemorrhage is doubtful. Even if total absorption occur permanent injury to the eyesight is still to be feared, especially after considerable hemorrhage in the eye and when the retina is detached.

(C) Abdominal and Thoracic Injuries

Since the delivery of the trunk rarely causes much difficulty, injuries of the internal organs are very uncommon, these being well protected from injury owing to their situation in the thoracic and abdominal cavities. The majority of hemorrhages which occur in thoracic and abdominal organs in the newborn are due to obstructed circulation. They may be increased in difficult deliveries by careless manipulation of the child or by attempts at resuscitation. We do not, however, recognize as true birth-injuries either the sub-serous extravasations in the liver or the suprarenal hæmatomata, &c.

Injuries of the lungs are very rare; they are only found after rib fractures which are themselves uncommon and only very exceptionally after the usually harmless fractures of the clavicle. In this case also the injury is more likely to take place after than during birth. Ruptures of the liver may occur when during an extraction from breech-presentation great pressure is exercised on the abdomen (Wallich, Knitter). The same applies to ruptures of the spleen and kidneys (Ballantyne, Smith).

In a child who died from hemorrhage into the posterior cranial fossa, in an asphyxiated condition, and in spite of attempts at resuscitation, the author observed a hemorrhage into the peritoneal cavity after a rupture of a connecting strand of peritoneum between the spleen and the abdominal wall.

Ruptures of the intestine, particularly of the large intestine, have been observed several times, but it is very improbable that these are birth-injuries (*vide infra*). Ruptures of liver and spleen may also occur before birth through injury of the mother (Geill).

PART VI

Diseases of Individual Organs and Systems

CHAPTER I

THE DIGESTIVE TRACT

(A) Diseases of the Buccal Cavity and the Jaws

INSPECTION of the mouth and throat is not very easy in the newly born. The child usually shows a definite resistance to the opening of the mouth; but if the spatula be pushed in as far back as possible between the jaw bones, these may usually be separated without difficulty; even when the mouth is open the narrowness of the cavity makes inspection difficult, especially that of the tonsils and pharynx. Perquet's fenestrated scapula is recommended even for the newborn child, instead of the solid form, as it can easily be directed perpendicularly to the cleft of the palate between the jaws and seen through with the assistance of the lighting arrangement.

The buccal mucous membrane of the newborn, especially that of the palate, shows great variations in vascularity. Sometimes it is pale pink and sometimes deep red. Erythema of the buccal mucous membrane resulting from hyperæmia may be looked on as a physiological condition in the newborn similar to erythema of the epidermis. The water losses of the first days are shown by the condition of the buccal mucous membrane which is usually fairly dry. It sometimes happens that the papillæ on the tongue stand out very noticeably. In correspondence with the desquamation of the epidermis which follows erythema, a thin coating is often found on the second or third day and pools off during the following days.

Very often minute extravasations of blood are to be seen during the first days in the mucous membrane of the palate or there may be more marked injection of the vessels, which gives a bright red and sometimes violet coloration to the palate.

If the mouth of a newly born child be inspected, whitish or yellowish nodules will be seen in or near the raphe of the palate, which in size and appearance resemble fine specks of gravel pushed under the mucous membrane. They occur in about the middle of the palate, usually in groups of three or four (fig. 43). Some-

times similar formations may be seen in the gums. These, which are known as Bohn's nodules or epithelial pearls, are to be found during the first days of life in about 90 per cent. of all newborn infants and are small retention cysts of the mucous glands. They usually disappear spontaneously, often after their upper layer has been torn, thus taking place without any operation being performed on the buccal mucous membrane. Sometimes small superficial ulcers arise, occasionally covered with a delicate coating and usually healing without any complications.

SPOTS ON THE PALATE; PERYGLOTTIC ULCERS (BEDNAR'S APHTHÆ). "SEPTIC" DISEASES OF THE MOUTH.

In the first days of life curious irregularly shaped spots frequently appear on the palate. They are sometimes circular, sometimes more jagged and lie either near the raphe on the boundary between the hard and the soft palate, or near the corners of the palate on one or on both sides. Originally delicate greyish-white with indistinct margins, they soon assume a more definite form. Their margins are then bright red and sometimes hæmorrhagic. The mucous membrane in the centre of the spot is anæmic, often yellowish as the result of commencing icterus; on closer examination a slimy skin-like layer may be seen which may easily be removed with a spatula; on the anæmic mucous membrane, which has been exposed, small punctate hæmorrhages may sometimes be seen. These spots on the palate are not visible on the first day, and do not, as a rule, appear until the second to the fourth day, they increase at first in intensity and then rapidly fade away. They have usually disappeared entirely by the end of the first week. The phenomenon is usually perfectly harmless. It has not been disproved that these spots may now and then cause pain during suckling, but as a rule they do not appear to cause any difficulties. The possibility of an infection is naturally not excluded. It has not quite been ascertained to what this loss of substance is due. It is probably connected with the wiping out of the mouth which is usually done by the midwife immediately after the birth of the child, even before the ligation of the cord, the object being to remove the mucus which has got into the mouth of the child and on to its palate during its transit through the genital canal. This procedure is certainly advisable with asphyxiated infants, but is perhaps not always necessary with normal children. If the wiping out is undertaken with a piece of sterile gauze and if the mouth be subsequently spared any further cleansing the child should not, as a rule, suffer any ill-effects.

That very slight lesions of the buccal mucous membrane may form portals of entry for bacteria is shown by the observations of K. Mayer, who saw an abscess develop in the angle of the jaw of a child who had fed from a breast affected with mastitic abscess, without there being any recognizable lesion of the mucous membrane.

While the aforementioned transitory spots on the palate appear during the first days of life, the periglotic ulcer (palatal angle

ulcer, Bednar's aphthae) which has been known for a long time, usually appears somewhat later. It may even be found in infants one to two weeks old. The pterygoid ulcer in its typical form is a spot about the size of a lentil which usually occurs symmetrically on the two sides near the hamulus. It is usually elongated and oval, and, as a rule, surrounded by a red border. It is the result of superficial infiltration, which rapidly ulcerates the surface forming mycotic necrosis of the epithelium, which, as Epstein has shown, occurs from the mucous membrane being easily injured and infected in vulnerable places, by the daily cleansing of the mouth. Sometimes a similar elongated area of loss of substance occurs in the raphe (fig. 46). Should the lateral ulcers enlarge towards the middle line they may unite with the ulcer on the raphe; in this way a butterfly shape results.



FIG. 45.—Bednar's nodules.

The treatment of palatal ulcers consists in gently touching them with a swab dipped in 2 per cent. caustic solution, or careful disinfection with H_2O_2 . The most important measure, both prophylactic and therapeutic, is prohibition of the daily "cleaning," a procedure which is quite superfluous in the toothless infant, as in spite of much care it is never without danger on account of the delicacy of the epithelium.

Uncomplicated pterygoid ulcer when treated in good time



FIG. 46.—Ulcers pterygoidei.
(Bednar's aphthae.)



FIG. 47.—Epstein's pseudo-leukoplakia.

permits of a favourable prognosis, but if it be not noticed and if the daily wipings continue, the inflammatory process may spread and lead to widespread necrosis of the epithelium and of the mucous membrane and also to the formation of deep ulcers with a pseudo-membranous deposit (Brecel). The disease known as "Epstein's

pseudo-diphtheria" develops in this way (fig. 47). It consists in greyish-white deposits resembling diphtheria on the posterior part of the hard and soft palate. On the virulence of the bacteria present in the mouth and on the resistance of the child depend the ultimate healing of the infection, or the development of the following complications: Severe septic stomatitis with purulent, necrotic inflammation or hæmorrhage; a septic inflammation of the nasopharynx and accessory sinuses; a phlegmon of the œsophagus and of the stomach (Linszenmeier), a localized erysipelas or a general septic infection arising from the injured mucous membrane (in which case there may be no severe local symptoms). Even mild affections of the mouth may exercise a very harmful influence on the welfare of the child, since nursing may be made painful and the intake of nutrition thereby reduced.

Finkelstein considers that the importance of affections of the mouth in young infants, which were exceedingly common before the discovery of antiseptics, is still very considerable. He states that out of one hundred infants who, when admitted to his institution were suffering from severe stomatitis, 24 per cent. died during the first three months of life, the cause of death being certainly or highly probably associated with the infection of the mouth. In the same period the death-rate of infants admitted with healthy mouths was only 8½ per cent. Finkelstein attributes this condition to the fact that in many maternity institutions in which the importance of asepsis in the treatment of the umbilicus is realized the danger of cleaning the mouth is either unknown or underestimated. All paediatrists are now convinced that the severe affections of the buccal cavity of the infant and their serious results may be avoided if the daily "cleaning" of the mouth which may convey infectious material from child to child be omitted.

Although the malpractice of daily cleansing of the mouth is still not rooted out, severe septic affections of the mouth (according to the records of earlier times terribly frequent results of originally harmless excoriations, catarrhal or parasitic stomatitis) are now among the greatest rarities not only in maternity hospitals but also in the large out-patient departments of children's hospitals in which people are treated who are not always characterized by particularly careful attention to hygienic principles. The improvement may be connected with the training of midwives in asepsis.

ORAL FOCUS OF STOMATITIS.

If the buccal cavity of the newborn be examined for bacteria anæmolytic staphylococci are chiefly found, such as may always be detected in the vagina of the mother. Sometimes streptococci may also be found, in which case it will also be possible to find them in the vagina of the mother. Bacilli, which are almost always present in the alkaline buccal secretion, are usually absent in the child's mouth, possibly because they are overgrown by cocci on the acid reaction which takes place after the intake of milk. *B. coli* is but rarely found (Neck, Kneise). The air saprophytes,

which have been found in the mouth of the newborn, probably have no pathological properties (Campa).

Although the cocci present in the mouth, especially the streptococci, have considerable importance in the genesis of alimentary infections, they very rarely cause stomatitis, if mechanical lesions of the mucous membrane are absent.

Catarrhal stomatitis in its pure form with swelling and increased secretion of the mucous membrane, is rare in the newly born child; as a rule it only accompanies the previously mentioned defects in the epithelium and thrush. Aphthous stomatitis and the ulcerative variety hardly ever occur in the newborn child or in any toothless infant.

A few cases of gonorrhoeal stomatitis have been described in the literature of the subject. According to Rosinski it takes the following form: "In certain places on the buccal mucous membrane whitish deposits occur: on the anterior two-thirds of the tongue, on the palate, in the region of the hamulus, along the pterygo-mandibular raphe down to the mandible, along the free margins of the jaws, in the raphe, and also in the gingivolabial fold of the mandible. The white coloration appears without any preceding inflammatory reddening. After twenty-four to thirty-six hours the colour becomes yellow, the spots become raised above the surrounding tissue, their surfaces becoming raw. The covering epithelium together with the extravasated pus cells forms a thick layer. The disease, of which the first signs are perceptible five to six days after birth, permits of a favourable prognosis. Healing begins even after a few days, an area of inflammation appearing round the patch and fresh epithelium grows under the yellowish mass. No scar or discoloration remains." Since in the cases which have been observed up to the present only extra-cellular "gonococci" were found and no cultures were made to verify the diagnosis, the question of gonorrhoeal stomatitis urgently demands revision. The opportunity for the occurrence of the disease is certainly afforded during the passage through the vagina of a gonorrhoeal woman. Also post-partum infection might take place from a conjunctival blennorrhoea. In a child suffering from this complaint Leyden observed on the upper lip the appearance of a yellow pustule which contained gonococci.

"Erythematous marginal glossitis" (Wertheimber) consists in reddening, increased prominence of the papillae and partial denudation of the epithelium on the lip and margins of the tongue. The latter swell and become sharply defined owing to their redness. It is said that the affection only occurs in artificially fed infants. As a rule speedy regeneration takes place after about a week. Wertheimber thinks that the process is a harmless one and is a physiological manifestation due to mechanical stimulation caused by sucking. Mom looks upon it as a traumatic product caused by the wiping out of the mouth. There seems to be no reason for assuming the existence of a definite disease.

Thrush. *Oidium albicans* settles chiefly in the mouths of infants whose general condition is in some way below par. It accompanies disturbances in nutrition in the newborn and especially septic infections. In such infants the tongue, mucous membrane of the cheeks and the inner surface of the lips are seen to be covered more or less thickly with thrush sores, these being frequently associated with Bednar's aphthae. That thrush itself can be the cause of a severe general disease is very improbable even though it may extend to the oesophagus or spread to the blood stream. The rôle played by thrush in the pathological condition is always secondary and its significance chiefly symptomatic. The occurrence of thrush in the mouth of an infant need not always be considered as an indication of a severe general disease or a greatly diminished power of resistance of the organism. In healthy breast-fed infants widespread thrush is undoubtedly uncommon, though little spots and superficial eruptions on the tongue or cheek are not so very rare. Stooß and Finkelstein are of opinion that the mouth of a perfectly healthy child will not tolerate thrush and also remains so when thrush elements lodge on it or when such are purposely inoculated into it (Epslein), but the meaning of the word "healthy" is used in too narrow a sense in this connection, at all events, for practical purposes.

Infection with thrush only takes place where the epithelium is damaged. Wiping out the mouth is therefore also of importance in the aetiology of thrush. *Oidium albicans*, when it reaches the mouth of the newborn child must usually have come from the nipples, unless by the use of dirty rags for wiping out the mouth this very common fungus is brought to the mouth from elsewhere and inoculated. Nosek points out that thrush may also originate from the mother's vagina, since *oidium albicans* is a frequent inhabitant of this tract.

Treatment consists in washing out the mouth with a 2 per cent. caustic solution or 25 per cent. borax glycerine; good results may also be obtained with Escherich's boracic sucking bag. Prophylactic measures consist in forbidding the wiping out of the mouth, and in keeping the breast clean. If the general hygienic principles of the puerperium and infant nursing be carefully adhered to thrush should not occur.

GANGRENOUS INFLAMMATION OF THE DENTAL PULP.

Under this name Svoboda describes a rare disease of early infancy previously known as *odotogingivitis gangranosa neonatorum* (Klementowsky). The six cases observed by Svoboda and Klementowsky fell ill on the 5th, 6th, 13th, 21st, 38th and 46th day; two of Zarfl's cases showed symptoms of disease during the second week. The most prominent symptom which in each case indicates a septic infection is the gangrenous destruction of the gums and the subsequent shedding of the crowns of the teeth. The disease follows one of two courses:

(1) The gums over the diseased tooth pulp are completely destroyed. In this case waxy discoloration of the parts takes place, separating them sharply from the surrounding tissues, their connection with the jaw gives way and the mortified part wobbles when touched. It then becomes gangrenous. The loosened crowns of the teeth appear out of their normal positions surrounded by pus and a gangrenous pulpy mass in the exposed alveolar fossae (in the newborn child the mammary surfaces of the milk teeth are on a level with the alveolar margins). The crowns of the teeth then fall out.

(2) The gangrene of the gums begins with a tumid livid swelling, then an ulcer forms over a tooth; from this a bloody and purulent exudate is discharged. The gangrenous destruction of the gums proceeds until, just as in type 1, it reaches the level of the mucous membrane, that is, until the loosened crowns which have been raised to the level of the mucous membrane or the edges of the ulcers are exposed. These crowns then fall out.

The disease begins with a protrusion of the gums similar to that occurring in normal dentition before a tooth is cut. It is not usually associated with great salivation and does not, as a rule, seem to cause much pain. The child's power of sucking is, however, always greatly reduced as it may cease altogether and the incapacity to suck is usually the first symptom noticed by the mother. The most striking symptom is that one day the doctor is presented with the crowns of teeth which have fallen out.

The prognosis of this disease must be described as unfavourable. The cases observed up to the present almost all ended fatally after no more than four to eight days; only in one case (Zurfl) did recovery take place.

TONSILLITIS.

Affections of the palatal tonsil and sore throat are among the rare occurrences in early infancy and throughout infancy the child is usually spared these common complaints of childhood. This may be due to the nature of the lymphatic apparatus of the pharynx. In comparison with the pharyngeal tonsil the palatal tonsil is only of secondary importance in early infancy. This is shown by the clinical fact that catarrhal inflammation of the roof of the pharynx is very frequent in infancy. Kretz was able to prove by anatomical investigation that in the infant the lymphatic glands at the angle of the jaw or those associated with the pharyngeal tonsil, are particularly plentiful in comparison with the glands of the mandible. Both local infections and widespread general infections spreading from the lymphatic organs of mouth and pharynx usually start in the young child in the palatal tonsil.

Eross observed follicular tonsillitis within the first seven days of life, which took the form of a transitory eruption of whitish, then yellowish spots on the palatal tonsil; there was no disturbance in the general condition. The rapid and transient rise in temper-

raure which Krüss observed in several cases cannot definitely be shown to be in causal association with tonsillitis as such. Transitory feverish attacks are so frequent at this period of life.

SWELLINGS WITHIN THE BUCCAL CAVITY.

Enlargement of the tongue, or *macroglossia*, depends either only on an increase in interstitial tissue (*macroglossia* of idiots, mongolism) or on true tumour formation. In the latter case it is a question of a vascular tumour which has affected certain portions of the tongue, of *haemangioma* or *lymphangioma*. Circumscribed lymphangiomas form small tumours about the size of a hazel nut, which are situated on the surface or back of the tongue showing a verrucose papillary surface (Parisch). Congenital cysts of the tongue (Cousins) and sarcoma (Marion) have been described.

Massin and Pith have observed a rare type of congenital tumour in the mouth. It was a connective tissue perithelial tumour of the gum of the upper jaw and was certainly benign, but owing to its size it interfered with sucking and had to be removed.

Serous cysts of the cheeks are situated between the corner of the mouth and the masseter and are covered with skin and mucous membrane, they appear to be painless and gradually enlarge. Congenital cysts and dermoids at the base of the tongue may cause the occurrence of laryngeal stridor.

ANOMALIES AND MALFORMATIONS OF THE MOUTH AND OF THE JAW.

The so-called *ankyloglossia* may be mentioned as the most common "abnormality," it is however an anomaly which only deserves to be classed as such in very rare cases. In the newborn child the frenulum linguae is normally attached directly behind the tip of the tongue; sometimes it reaches right to the tip and appears to be inserted into a depression in it. There are great variations in the length and insertion of the frenulum without any anomaly being involved; *ankyloglossia* represents at least to a certain degree a physiological stage in the development of the tongue of the newborn child. That "tongue-tie," so often diagnosed by the laity, may be a hindrance to sucking, is shown not to be the case, because during sucking the tongue is not moved backwards and forwards but upwards and downwards. Division of the frenulum which is so often desired by the mother in older infants is an unnecessary but harmless procedure, and in the newborn child best omitted. It must be explained to the mothers that their fears are groundless.

A rare malformation of the tongue is a median cleft (*lingua bifida*).

The rare circumstance in which teeth are present at birth is known as *dentito praecox*. It is usually the two lower incisors which appear either fully cut or as cartilaginous lumps covered by mucous membrane projecting from the alveolar margin; only quite

exceptionally is the number greater (8, Oriola). The teeth are liable to cause difficulty to the mother during suckling. Should this be the case they must be removed, this being easily done with small forceps. Otherwise the teeth usually fall out of their own accord in course of time or they may be shed owing to inflammatory symptoms in their neighbourhood.

The following anomalies of the jaws are known: micrognathia, abnormal shortness of the mandible, usually associated with a small tongue; polygnathia or epignathia, duplication of the mandible which takes the form of a swelling covered with mucous membrane, external to the normal mandible (Mayer); agnathia, absence of the mandible, usually associated with other malformations, defective development of the maxilla, absence of the mouth opening, cyclopia, &c.; hemignathia, absence of lower half of the mandible (Bürger).

Much more frequent and of greater importance than these malformations are the congenital clefts in the facial skeleton, hare-lip (lateral cleft of the upper lip, *labium leporinum*, *cheiloschisis*) and cleft palate (*uranichisis*, *palatoschisis*).

Hare-lip occurs in very variable degree and type. It may be limited to a slight furrowing of the lip; in other cases the cleft reaches right to the nostril. The red part of the lips is either not cleft or it merges into the nasal mucous membrane. Hare-lip is not infrequently associated with clefts in the process of the superior maxilla and of the palate (*cheilognathopalatoschisis*). The cleft may be unilateral or bilateral.

The palatal cleft also may be only on one side of the vomer or it may be on both. The cleft may affect the maxilla, the hard and the soft palate, or it may be limited to the latter and to the uvula. Should it affect the palate alone, it is spoken of as a *uranocoloma*.

We will not deal further with the details, the genesis, or the operative treatment of these malformations. That an operation is necessary, if only on æsthetic grounds, is obvious. The most important question is to decide when to operate. Spitzzy recommends that hare-lip should only be operated on when the child has reached a weight of more than 3,000 gm. and is in a healthy condition; earlier operation is only indicated when the malformation is so extreme that it would in itself prevent the continued life of the child. Cleft palate should not be operated on before the end of the first year.

Very considerable difficulty is sometimes met with in the feeding of infants with large clefts of the lip and palate, since they may be unable not only to take hold of the nipple well, but also to suck effectively, they are also liable to choke. Should sucking direct from the breast be found impossible—the attempt should not be abandoned too early—bottle feeding must be substituted; this does not, of course, mean that artificial feeding should be resorted to, but that pumped-off milk should be used. It is well to push the teat far back into the mouth.

Another and much rarer facial cleft is the oblique cleft, which runs from the mouth or nose towards the eye, and which may be combined with clefts of the eyelids. There is also a transverse cleft of the face which results in a widening of the mouth towards the ears.

Congenital cervical fistulae are divided into lateral fistulae, which result from defective closure of the second branchial cleft, and median fistulae which are due to the persistence of the thyroglossal duct. While the median fistulae are usually secondary occurrences, that is, not congenital, the lateral are usually present at birth. The external openings of the fistulae may be situated in various parts of the side of the neck; the inner opening is placed in the tonsillar region near the palato-pharyngeal arch or on the lateral wall of the pharynx. The fistula may be complete or incomplete, according to whether it is permeable or not; an incomplete fistula may be external or internal. The canal of the fistula may usually be felt through the skin like a cord. The secretion is a mucous fluid, sometimes clear and sometimes turbid. The cysts may also be formed secondarily owing to the breaking down of a cystic tumour in the neck (cyst of a germinal duct).

SUPPLEMENTARY NOTE.

Lymphangioma (Hygroma). Colla Cysticum Congenitum.

Congenital cervical hygroma appears as a large swelling situated laterally in the neck near the great vessels. It shows fairly rapid growth, and may, after a time, reach not only to the clavicle, but may pass below this bone, pressing into the thoracic muscles, and eventually causing difficulties in respiration and swallowing owing to pressing upon the trachea and oesophagus; death from asphyxia may even result. The tumour in question contains many cavities, is cystic and filled with clear lymph, and it is usually closely adherent to the surrounding tissues. For this reason radical removal is very difficult, but is nevertheless the only treatment which offers a ray of hope. Recurrence is to be feared after operation (Spitzzy).

(B) Diseases of the Salivary Glands

(1) CONGENITAL ANOMALIES AND DISEASES.

Defects of individual glands (submaxillary, Grüber) and anomalies of position have an anatomical rather than a clinical interest, and usually escape observation during the newborn period. The same applies to the rare cases of abnormal opening of one of the ducts (congenital salivary fistula).

Salivary stones occur, although exceedingly rarely, even in earliest infancy. Bardet removed a stone from the submaxillary duct of a child aged three weeks. This was about the size of a grain of wheat, and must have been present before birth. Cloquet

mentions a case of the congenital formation of concretion in the sublingual duct. The stone was as large as a barley corn, and consisted chiefly of calcium phosphate. The sublingual gland was greatly swollen and interfered with sucking; on pressure, one end of the stone protruded from the orifice of the duct. The stone was removed by forceps without incision.

Congenital cysts of the salivary ducts are caused by obstruction of the duct by a stone or congenital imperforation. Sultan observed a case of bilateral congenital impermeability of the submaxillary duct which led to the formation of cysts the size of a plum stone on the floor of the mouth.

They were present from birth, were filled with transparent contents, and projected with a curved horn-like process into the mouth. A cyst of the duct of Blandin and Nuhn's gland forms a transparent vesicle at the tip of the tongue or just under it, and may project between the lips. As a rule such cysts do not attain to any great size. They finally remain stationary or perforation occurs through the duct or as a result of infection in some other place. In a case of Johnson's a cyst of the sublingual gland, present before birth, interfered with respiration and made tracheotomy necessary after four months. If a cyst interferes greatly with breathing or sucking, operative interference cannot be avoided. This is carried out as follows: As near as possible to the natural orifice of the duct a needle threaded with silk is pushed in through the mucous membrane and the cyst, emerging again a certain distance on the opposite side. The ends of the thread are tied tightly, so that the ligatured bridge becomes necrotic and a salivary fistula is formed which functions as a normal orifice. Another method is to open the cyst and to excise a portion of its wall. The edges of the mucous membrane and the cyst are sutured together (Kunier).

Ranula also occurs in the newly born. It is probably due to a retention cyst of the sublingual gland which fills the space between the frenulum linguae and the mental portion of the mandible, and it may push the tongue upwards. Operative treatment made necessary by interference with sucking must proceed very carefully, as after mere opening of the cyst recurrence takes place.

Kien describes congenital enlargement of the parotid. It appears as a large, regular turgid swelling of the parotid gland without any inflammatory symptoms; in the course of the first weeks of life it gradually diminishes in size. Since in neither of the cases observed by Kien was there any occlusion of the duct, retention of saliva cannot be the cause of the enlargement. It probably owes its existence to interference with the venous return on account of the long duration of labour. Perhaps it is comparable to the congestive swelling of the thyroid gland which also disappears gradually and of its own accord.

Congenital tumours of the salivary glands are rare. Haemangioma of the parotid is relatively the most frequent and important. It is usually noticeable from the first day of life onwards, or tends to

appear shortly after birth. Though pathologically a benign tumour it is by no means clinically harmless. It takes on a rapid growth, so that a tumour, at first the size of a nut, develops within a few months to such an extent, that it may involve both cheeks and neck; it may lead to atrophy of the jaw, and even finally result in symptoms of suffocation from pressure on the larynx. Untreated cases, hitherto observed, have all succumbed. The radical removal of the tumour must therefore be resorted to, and in view of its rapid growth, the danger of hæmorrhage and a lesion of the facial nerve, the operation should be undertaken as early as possible. With correct treatment the prognosis is favourable, for after total extirpation recurrences do not recur. As a rule, the tumour is more or less compressible, its size increases on crying, and its bluish colour shines through the intact skin (Duplay, Lewitz, v. Haberer, Hartas and Suchter).

Lymphangioma of the salivary glands generally appears to run a benign course. Hagenbach describes a case of symmetrical lymphangioma involving all the salivary glands. Lipoma (Sauri), sarcoma (Göthe) and adenoma or cystadenoma (Eröss, Herleimer, Sacchi, v. Saar) have also been observed in the parotid gland. Braque-Haye and Sobrazes describe a case of bilateral tumours of the sublingual salivary glands, present since birth. According to the opinion of these authorities it was not a formation of true adenoma, but of "macroadenia."

(2) INFLAMMATION OF THE SALIVARY GLANDS (ACUTE PURULENT SIALO-ADENITIS).

Purulent inflammation of the salivary glands, a rare disease, is observed relatively frequently just at this early stage of infancy. Purulent sialoadenitis does not only attack the submaxillary and sublingual glands, as assumed by Hennig, Mikulicz and Kimmel; according to the cases hitherto published, the parotid gland seems to be that most frequently involved (Nobécourt-Hutinel, Hofstaetter, Aurbach, Breitschneider). In most cases the causes of the disease are staphylococci. Perrot found in one case streptococci, and Brenden nitium albirans, besides staphylococci.

The disease generally occurs in the course of the first or second week, though it has repeatedly been observed at a later period. It does not appear to occur before the third day. It attacks either one gland (as a rule the parotid, more rarely a submaxillary gland) or several (both parotid glands, or those at the floor of the mouth, or both together). If several glands are involved they are usually attacked in succession. The first striking symptom is the swelling. It is situated according to the gland, either over the cheek, in front of the ear, at the angle of the jaw, or below the horizontal ramus of the lower jaw. The skin is generally at first unchanged, fluctuation at the beginning of the disease cannot usually be found. But in the early stages, pus will come out of the excretory ducts by pressing on the swollen parts, that is on the buccal mucous membrane

above the alveolar margin of the upper jaw, where the latter bends backwards, and at the plicæ sublingualis or frenulum lingue. The mouth of the excretory ducts may be reddened or somewhat dilated. The temperature is very variable. The disease may sometimes pursue its course with very high fever, in other cases this may be slight or exist merely during the first few days. An afebrile course may also occur, particularly in debilitated children. The other general symptoms and signs vary in isolated cases. The children are either limp and refuse nourishment, or feeding is in no way affected and the general state of health seems to be little disturbed. A certain tenderness of the glands affected is always present.

The course and prognosis depend largely on the constitution and powers of resistance. Relatively frequently the disease attacks premature infants (according to Bretschneider 40 per cent.) and these generally succumb to pyæmia, sepsis or ecchæmia; from this point of view the prognosis is, on the whole, most unfavourable, but with correct treatment, strong and otherwise healthy children may recover, even spontaneously. Among complications, apart from general infection, the spreading of suppuration in the neighbourhood is that most likely to be feared; penetration into the external auditory canal, burrowing of pus along the cervical vessels into the mediastinum. Facial paralysis is also liable to occur. Serious intestinal affections through swallowing of pus containing cocci, practically never occurs. This coincides completely with our experiences concerning the relatively slight danger of sucking at a breast with mastitis.

The pathogenesis of the disease is disputed. On the analogy of metastatic parotitis, which occurs during the course of infectious diseases (typhoid), glandular suppuration in the newly born has been stated to be associated with general sepsis and attributed to a hæmatogenous infection (Perot). Though this statement may hold good for a few isolated cases, for the majority the direct passage of infection is more probably by way of the excretory duct of the gland. Pus cocci are always found in the oral cavity or on the skin of the nipple. The co-existence of puerperal diseases and fissures of the nipple may heighten the possibility of infection. Stomatitis need, by no means, always be present, but is more likely to be secondary. Why the ubiquitous cocci so seldom wander into a glandular excretory duct needs an explanation. Hofscatter thinks that a crushing of the infantile parotid gland by pressure of the obstetrician's hand on the face when extracting the shoulder through the rigid soft parts of the mother, may also be a cause of later infection. The disease has frequently been found with forceps deliveries. Possibly a deficient disposition or functioning of the protective mechanism, or a deficient bactericidal action of the saliva may also be responsible. There may be retardation of the secretion resulting in a diminished stream of saliva (Bretschneider).

Treatment at the beginning of the disease should be confined to antiphlogistic compresses (lead subacetate lotion). It is also

recommended that the purulent secretion be removed by regular pressure of the gland, and the mouth, in particular, the region of the duct orifice carefully wiped out with disinfecting fluid (boracic solution, H_2O_2). Most authorities advise early and even numerous incisions at various parts of the infected region, also, where the tense fascia parotideo-masseterica interferes with the sensation of fluctuation (Baum).

As with every disease of the newborn child, the nourishment must be, if possible, the natural one. In many cases suckling was continued, in spite of the disease, without any infection occurring to the maternal breast, though galactophoritis has been observed at the same time (Lequeux). It would therefore be advisable not to put the child directly to the breast, but either to let it suck through a nipple shield or to feed it with milk drawn off.

For parotitis epidemica, *vide infra*.

(C) Diseases of the Gastro-intestinal Tract

During the last decade a change has taken place in this section of infant's diseases, in that in place of the narrower term, "disturbances of digestion," "disturbances of nutrition" has been substituted. The cause for this lies chiefly in the clinical observation that children, whose nutritive functions are seriously affected, by no means always show severe symptoms of disturbances of digestion in the narrower sense, *viz.*, local disease of the gastro-intestinal canal. On the other hand, obvious signs of intestinal disease, *e.g.*, intestinal catarrh, enteritis, may exist without the children in question being essentially affected in their general condition of nutrition. Furthermore, we have learnt to recognize that the primary cause of disturbance of nutrition is not always found in disease of the intestinal canal, the organ primarily concerned with digestion; but that it is primarily the lowered power of resistance of the whole organism from general disturbances of nutrition which is the cause of involvement of the intestine. In this manner the symptoms of an intestinal disease may be of a secondary nature. The term "disturbances of nutrition" covers in its scope not so much those disturbances of the general condition, which are caused by diseases of the intestinal tract or faults in nutrition, but it embraces far more those general disturbances which are caused by the fact that the general processes of nutrition, including digestion, absorption, assimilation, internal decomposition, and intermediary processes do not occur normally.

Czerny and Keller, to whom we are indebted for the first aetiological division of disturbances of nutrition according to the latest theories, classify them according as they are due to feeding, infection or constitution. Strictly speaking, only disturbances of nutrition due to infection come under consideration during the newborn period, and, in fact, in so far as they are included in enterogenous sepsis. Both the other forms only become manifest after a certain period of latency, and therefore hardly come under

the category of diseases of the newly born. If further details of this subject are not discussed here, it must nevertheless be strongly emphasized that the first and most important injuries leading to that which is known as disturbance of nutrition often attack the child just in this very first period of life, and that the prophylactic treatment for by far the most important diseases of the newborn period falls to the lot of those in charge of the feeding and nursing of the baby from the day of its birth. It must be taken into consideration that of the constitution and power of resistance of the child (unless premature or obviously debilitated) at this time we have no distinct clinical knowledge, and in any case the generally recognized optimal conditions must be accepted. As it cannot be known how a child will react to artificial feeding, even if this shows apparently good results in the first two to three weeks, energetic steps to maintain breast-feeding must be taken, and in the case when there are contra-indications on the part of the mother to suckling, it must be seriously deliberated whether the cessation of breast-feeding may be possibly of greater detriment to the child than it is of advantage to the mother. The deceptive latency in disturbances of nutrition (due to feeding and constitution) during the newborn period is unfortunately a frequent ground for this common and thoughtless resort to artificial feeding. If the latter cannot possibly be avoided, it must be most carefully carried out. So long as the infant comes under the category of newly born, our primary effort must be to preserve the intestinal tract as far as possible, even if the increase in weight leaves at first much to be desired. For the significance and avoidance of infectious noxae, see p. 488.

Whereas the real disturbances of nutrition tend to be latent during the first period of life, local diseases of the gastro-intestinal canal play an important part in the newly born.

VOMITING.

Vomiting is a symptom and not a disease. If it is discussed here in a section by itself, this is due to the fact that it is generally difficult at first to decide with any certainty what is the cause of the vomiting. A classification according to primary diseases would not correspond to clinical requirements; the medical man must know before anything what may be the causes of vomiting, and what importance must be attached to the symptom and how to treat it.

That form of vomiting which occurs from obstruction to the lumen of the digestive tract is usually easy to diagnose from the other symptoms; also septic vomiting, and hæmatemesis occurring with mælena, require a special place.

In the course of the first day many children vomit liquor amnii, vaginal secretion and blood swallowed during birth. The vomit accordingly consists of either colourless fluid, combined more or less with mucus or blood-stained brown-coloured substances. The vomiting may be repeated two or three times and reach considerable

proportions. The retching movements shown by some children, as also the brown colour of the vomit, occasionally frightens the mothers terribly, and it is the business of the medical man or nurse to explain the harmless nature of the symptom, which must be regarded merely as a physiological one.

During the following days the children frequently vomit if the taking of food has begun. It may simply be the result of too large quantities of food; with a readily secreting breast a child may easily have, relatively speaking, too much to drink. But even with small quantities of food vomiting may occur. The children either vomit immediately after food, sometimes whilst still at the breast, or a little while afterwards, as soon as they are laid back in the cradle. In these cases the milk either runs in unchanged condition out of the mouth, or is poured forth, without the child showing any further symptoms of discomfort. In these cases there is apparently a deficient closure of the cardia. Possibly in some there may be an atony of the muscular apparatus of the stomach, in which the cardia participates; with compression of the abdomen, and thereby of the stomach, the closure of the cardia is easily overcome, and the contents of the stomach flow out through the œsophagus (Peiser). The tonus of the cardia in the young infant is relatively slight under entirely physiological conditions, and mere contraction of the antrum pylori suffices for the emission of the contents of the stomach without the abdominal muscular pressure being brought into action. In other cases vomiting only occurs after two to three hours, sometimes shortly before the next time for feeding. The milk is then curdled and has a distinctly sour smell; the vomiting reflex is possibly caused by supersensitiveness of the gastric mucous membrane. The regurgitation or vomiting of the food may occur once or several times a day, and may disappear suddenly or gradually during the following days.

Vomiting, as a rule, is only a passing symptom of the newly born and possesses but little importance. If it lasts for over a week, however, though varying in intensity and frequency, a considerable degree of under-nourishment may result. It is often difficult to decide which form of vomiting will develop, whether the generally harmless, the habitual, the uncontrollable, or the pyloro-spastic form (Finkelstein). Distinction between these can be made on the grounds of their sequelæ, which are shown according to the intensity of the vomiting in the general condition of the patient. In the spastic type the nature of the vomiting is at first not very distinguishable. According to Finkelstein, the difference between habitual and uncontrollable vomiting is merely one of degree. In the latter form the vomiting is so severe that inanition results and increase of weight is impossible. Symptomatically both forms are otherwise much the same. Ischydymia and severe fermentation cannot be found. When secretory anomalies are present they are confined to a mild catarrh (accumulation of mucus).

Guinon believes that vomiting in the newly born is sometimes

caused by the children swallowing air. Owing to furling of the nostrils a small cleft is left open at the lip commissure, through which air is sucked in. When breathing through the nose improves the swallowing of air and vomiting ceases.

Habitual vomiting, which occurs in many children during the first few months of life, is considered by many as a sign of nervousness or of a neuropathic constitution. In this respect other symptoms (restlessness, fright, &c.) must be taken into account, but before giving an opinion it is best to let the newborn period pass.

Vomiting in itself presents, as a rule, no danger to the child, even though under certain circumstances it may be very unpleasant, especially if the greater part of a scanty supply of milk from the breast is vomited and so wasted. In premature children with small reflex excitability or in debilitated children vomiting is far more serious, for aspiration of the vomit with its consequences may arise. In uncontrollable vomiting the danger of inanition and exsiccation come under consideration.

Treatment for vomiting.—Above all, the technique of feeding must be regular. The sucking power of the infant and the quality of the breast milk must be taken into consideration, the intervals between meals, and the number, amount and duration of the meals must be regulated. If, in spite of correct technique of nutrition the infant vomits, there is no need to worry at first, but the usual procedure should be continued. If the stools are not of the nature of starvation stools inanition is not to be feared. All manipulations of the child should be carried out as gently as possible, severe mechanical shakings should be avoided. In order to prevent the return of the stomach contents, the upper part of the child's body should be placed in an oblique position. If children disposed to vomit are in the habit of crying after their meal it is permissible to soothe them with a comforter. In the great majority of cases there is no necessity for more drastic measures.

If the vomiting assumes a more threatening aspect, one may try to soothe the affected stomach by occasional meals with tea or with mother's milk deprived of its fat. But, as a rule, the results of these measures are not very striking; some children, in fact, vomit more easily after tea than after milk. One may also try to reduce the size of the feed by shortening the period at the breast. If these simple methods are not successful an exact amount of fluid must be given, which is only possible by giving drawn-off milk in a bottle. This can be done, just as in vomiting of older infants, by giving quite small quantities (5 to 10 gm.), at short intervals (hourly), or cold and gradually increasing the amount at longer intervals until the child can be put once more to the breast. (In order to prevent the exsiccation which may so easily occur, water enemata or subcutaneous infusions must be given. The superfluous milk must be removed by pumping it off from the breast.) The latter method, especially with premature children who cannot suck at the breast, frequently yields excellent results.

Washing out the stomach may be tried, but not much success must be expected from it, as, with the usual forms of vomiting in the newly born, there is generally no stagnation necessitating the removal of decomposed stomach contents.

With regard to the medicinal treatment the administration of cocaine, recommended by Rott, must be mentioned. The author himself believes good results have been gained by it. Ten c.c. of a solution of cocaine hydrochl., 0.05 to 100 of water should be given for three days, five times a day and ten minutes before meals.

INTESTINAL DISEASES.

In contrast with the later period of infancy, the relatively easy permeability of the intestinal wall during the first few days of life (see p. 33) presents opportunity for many intestinal infections and intoxications. Pathogenic bacteria or the products of their metabolism (true bacterial toxins) may pass through the intestinal wall and give rise to enterogenous sepsis; so also normal intestinal bacteria may form toxic substances from the chyme, and possibly the intestinal secretion, which do not pass through the normally functioning intestine, but may occasionally pass through the ill-developed intestine of the newly born and produce toxic effects in the organism. Finally, products of decomposition of intestinal contents not formed by the activity of bacteria, but possibly by the physiological digestive juices, may get into the body and produce symptoms of disease. If an injury is added to the functional backwardness of the intestinal wall (either from chemical, physical-chemical, toxic, or bacterial influences), the opportunity is still more favourable for the passage of these toxic substances. In this manner possibly many a disease of obscure aetiology may be explained and the basis formed for many later "disturbances of nutrition." We certainly know of no sharply defined clinical pictures corresponding to the above aetiological factors. The functional inferiority of the intestinal wall can, of course, not be recognized. From the symptoms exhibited by the digestive tract, injuries can hardly be concluded, because that which strikes one clinically as "intestinal catarrh" or "enteritis" corresponds to diseases of the lower parts of the intestine, particularly of the colon; whereas the general consequences of injury to the intestine are all the more serious, the higher the portion of intestine involved, and it is just the disease of the upper part of the small intestine, so important in the processes of digestion, that is so very difficult to diagnose with certainty by the character of the stools.

Intestinal Catarrh (Dyspepsia).

While discussing the character of the normal stool, it was pointed out that milk stools which follow meconium practically never exhibit the character of the so-called "normal" breast stool. When the brown, slimy, "transitional" stools are evacuated, and when the yellow-coloured breast stools appear, somewhere between the

third and fifth day, they nearly always have the character of "dyspeptic" stools, and are slimy, crumbly, and more or less thin. The intestinal mucous membrane of the newborn child reacts under quite physiological conditions to the food with symptoms of irritation, and apparently particularly in the case of mother's milk which is rich in sugar. The irritants may be partly derived from the invasion of bacteria into the bowel, and may be partly of an alimentary nature; as in similar conditions of older breast-fed children, the acids due to fermentation through the activity of bacteria probably play the most important part. This transitional catarrh must be considered as physiological. When it is absent, and only scanty dark-yellow or brown stools, poor mucus and water, are passed, one is generally dealing with an underfed child. It is most difficult to decide in the so-called dyspepsia of the newly born where the boundary may be drawn between "physiological" and "pathological." The number of stools is the best guide. Three to four stools daily may be considered normal; five or more stools indicate catarrh.

The expression "dyspepsia," which has unfortunately crept into the most modern publications, is most undesirable. Dyspepsia, literally translated, means disturbance of digestion; but, on the one hand, a disturbance of digestion is not necessarily combined with symptoms of intestinal irritation; on the other hand, an intestinal catarrh of the lower portions of the intestine is scarcely ever combined with disturbance of digestion, in other words, the breaking down of food. The substitution of the good old word "intestinal catarrh" for the obscure term "dyspepsia" would be very desirable.

The symptoms of intestinal irritation readily occur in powerfully sucking infants of women with an abundant supply of milk, but are by no means exclusively due to overfeeding. Even with strict observance of five meals and with quantities not exceeding the normal amount they may often be seen to occur. The children evacuate from seven to eight, sometimes from ten to twelve stools daily. They are soft, pulpy, slimy, loose, sometimes spurring, very thin, sometimes of a light yellow colour, interspersed with crumbly fragments; sometimes combined with a glairy and sometimes with an opaque (cellular) mucus. At intervals quite good, homogeneous, oily, gold-coloured breast stools may be evacuated; occasionally such a "classic breast stool" may immediately follow a spurring evacuation. The stools assume a green colour in the napkin, especially when they have a strong acid reaction; they may also be green immediately they are evacuated.

One must always endeavour to see the evacuations immediately, as stools, rich in water, change considerably in appearance after being any length of time in the napkin. Besides the number of stools the amount of each evacuation must also be carefully noticed.

The infants are frequently very restless; they cry from colic or from intertrigo round the buttocks, which easily occurs. Severe general disturbances are mostly absent in the usual inflammatory catarrhs. It is hardly probable that feverish conditions can be

associated with them. Now and then the children also vomit, but this is apparently not a case of true gastric dyspepsia, but a casual coincidence, probably the result of relatively too abundant feeding.

Wherein lies the cause of this common intestinal catarrh? It is probably an increase of the physiological irritative catarrh, possibly from the formation of too much acid, possibly from an individually varying sensitiveness of the intestinal mucous membrane. It is improbable that the colostrous character of the early milk is the cause of the catarrhal symptoms, as many children show no signs of intestinal catarrh when fed with milk rich in colostrum.

It has been considered by many that one is here dealing with infective processes. Kermanner and Orth believe that the relatively unfavourable health of infants in many lying-in institutions, to which amongst other matters may be attributed the deficient increase in weight, might be caused by that dyspepsia which they regard as a uniform clinical picture, and attribute to staphylococcal infections. Even though it must be admitted, without reserve, that strict cleanliness in the feeding of the newborn child is most desirable for the prevention of intestinal troubles, and that in this respect there is much to be desired in many lying-in institutions, it is still apparently very doubtful whether intestinal catarrhs may be prevented by asepsis alone. It is probably impossible to prevent staphylococci reaching the oral cavity of the child and thence passing to the intestine, even if the precaution is taken of putting the children to the breast with a nipple shield, for the cocci may originate either in the genital passages of the mother or the excretory ducts of the breast. According to Schabert during the period of thirty-two hours post-partum up to the fourth day of life diplo- and staphylococci are invariably to be found in the infant's stools. The earlier the cocci appear the earlier the stools assume their dyspeptic character; they predominate at the height of the intestinal symptoms. Schabert therefore believes that every child must have staphylococcal enteritis during the first few days. The aetiological significance of these cocci is certainly questionable. Staphylococci or enterococci may come from the upper portions of the intestine and thereby be more abundantly present in the more frequent and rapidly evacuated dyspeptic stools, without having other than a secondary importance.

The author has frequently observed that newborn children usually at the end of the first week temporarily evacuate blood-stained, mucus stools, resembling dysenteric stools, without any further prejudice to their general health resulting.

The prognosis of the catarrh is generally entirely favourable. As a rule the stools improve spontaneously during the second or third week, both in their amount and quality. In other cases dyspepsia of breast-fed children is associated with these stools. In the most favourable cases the symptoms only tend to disappear after six weeks, in more severe cases after months, and not infrequently only after the administration of mixed diet. Apart from the rela-

tively rare cases, (characterized by failure to gain weight or insufficient thriving, or associated with anomalies of constitution (exudative, neuropathic diatheses, &c.) these intestinal catarrhs of breast-fed children are harmless, though extremely tedious diseases for both mother and medical attendant, on account of the colic, restlessness of the infant, necessity of most careful nursing, and also of the difficulty of the treatment of the infant. It therefore behoves one to be cautious with any prediction during the first week of life.

Treatment at first must be confined to what is urgently needed, viz., regularity of meal times, and later a limited supply of food. The children should still be put to the breast, as improvement generally occurs spontaneously and thriving is not as a rule prejudiced by the catarrh. The introduction of starvation diet (24-hourly tea diet) is, as a rule, not necessary during the first week and in general, not to be recommended. At the most the amount of meals may be limited by substituting feeds with tea (with saccharin) but the result of these measures is not very satisfactory. It is necessary to impress upon the mother that the bad stools are not caused by the inferiority of the mother's or wet-nurse's milk and that any other woman's milk would, in all probability, effect no change in the quality of the stools. If in isolated cases one imagines that the change of nurse has had a beneficial effect on the catarrhal symptoms, the cause of the latter lies nearly always with the child, either in its intestinal flora or in the susceptibility of its intestinal mucous membrane.

In breast-fed children with a very strong acid reaction of the stools, sometimes the additional feeding with cow's milk (poor in or free from sugar) or with casein-fat skimmed off, albumen milk, &c., has a beneficial effect on the character of the stools. In the first weeks this additional feeding is, as a rule, superfluous. Too much must not be expected from medicinal treatment. Administration of tannin preparations renders the stools sometimes drier. Tannalbin, tannigen, &c., may be given in doses of 0.25 gm. four to six times daily, best of all with a little drawn off milk in a spoon. In all cases these harmless methods may be recommended. Also the bacterial toxin and water fixation methods (Boi. alb. 30:0/100; animal charcoal 1 g. in 100 c.cm. tea) may be tried with small children. If it is a true "dyspeptic" case, a permanent effect is not generally secured by these methods. For colic any warm compresses on the abdomen (thermophor, linseed, baked camomile, &c.) often have an excellent effect. Whether carminatives, favoured by so many mothers, have really much alleviating effect is open to doubt, in any case they can be safely prescribed for newly born (camomile tea or aqua carminativa chamamille, dill water, &c., administered in teaspoonfuls).

It is a well-known fact among paediatricists that there are cases of persistent dyspepsia and insufficient thriving, which only show improvement when natural feeding is stopped and some form of feeding with cow's milk is resorted to—cases in which artificial feeding proves itself superior to natural. Nevertheless, it must be emphasized that these are exceptional cases, and that so

long as the child may be classified as newly born, say during the first two or three weeks, one is hardly ever justified in assuming such an exceptional case.

Enteritis and Enteral Infection.

Whereas the aforementioned intestinal diseases are probably chiefly due to the irritation of food and its products of decomposition, and bacterial products of decomposition only play a part, in as much as they arise from activity of the normal intestinal bacteria, there are doubtless also enteritides which are caused by pathogenic micro-organisms. These diseases, however, in which one is justified in assuming abnormal infectious processes to occur in the intestine, cannot be very sharply defined in the present state of knowledge. If one speaks of an infectious disease, one naturally inquires after the cause of the infection, and to this question the answer is often missing.

There can hardly be any doubt that in the intestine of breast-fed babies there are sometimes processes of putrefaction, without any pronounced symptoms of disease appearing. The chief representative of the characteristic meconium flora is the gas-phlegmon bacillus, a micro-organism which forms products of putrefaction on a nutrient medium containing albumin and no sugar (Passini). Possibly the not infrequent indicanuria occurring, on the third or fourth day, in quite normal breast-fed children, is associated with these kinds of enteral processes of putrefaction, even if, as a rule, no parallel can be drawn between indican excretion and the quality of the stool. It follows that during the first few days of life processes of reduction take place in the intestine (such as are generally combined with processes of putrefaction and correspondingly tend to be completely absent in the intestines of older breast-fed children). For at the beginning of the breast-stool period, even in yellow-coloured stools with a markedly acid reaction, there is sometimes a positive urobilinogen and urobilin reaction; and such stools sometimes show a red area of stercobilin in the napkins, which otherwise only occurs in stools after cow's milk.

If such symptoms indicate no abnormal processes in the intestinal tract, they remain, as a rule, without any clinical significance. According to Passini's researches the products of metabolism of the sporulating, septic, proliferating forms of the gas-phlegmon bacillus are less to be feared than those of fermenting forms which arise with the appearance of food containing sugar in the intestine. Sometimes during the first days of suckling, at the time of the so-called transitional stools, very frothy stools with much mucus are observed. According to Passini's theory it might be imagined that the arrival in the lower portions of the intestine, of milk remains containing sugar, creates a transformation of the sporulating proliferating form of the gas-phlegmon bacillus into its gas-forming asporogenic variety. As these fermentative forms of the bacillus tend to form poisonous substances *in vitro*, it is conceivable that they play some part in the pathogenesis of certain pathological

symptoms at the time of the transition of the meconium to the milk-flora. These symptoms would be cases of enteral auto-intoxications, caused by a normal intestinal microbe.

In contrast with the earlier mentioned, insignificant processes of putrefaction during the transitional period, cases have been observed in which the processes of putrefaction in the intestine gain considerable intensity and are accompanied by symptoms of disease.

Berend describes the following clinical picture: initial vomiting on the first day of life, before food; on the third to fourth day moderate, seldom high, fever and simultaneously, at the end of the meconium period, but seldom later, offensive, mucous evacuations. In favourable cases, recovery after one or two days, in more severe cases with conspicuous loss of weight, after ten to fourteen days; fatal cases have also been observed. Berend presumes that the cause of the disease is to be found in the swallowing of decomposed liquor amnii.

Cramer reports on two cases which he regards as septic infections arising in the intestine. In one case the child evacuated on the second day putrid-smelling meconium and died at the age of $2\frac{1}{2}$ days. The post-mortem showed swelling of the follicles, extending through the whole intestinal canal, a part of which had become ulcerated in the large intestine, and much swelling of the mesenteric glands. In the second case the child refused nourishment on the third day and died cyanosed. The post-mortem showed signs of follicular enteritis; injection and extravasations of blood in the mucous membrane of the small intestine; swelling of Peyer's patches and follicles of the large intestine; swelling of the mesenteric lymph glands. Cramer suggests infection with streptococci which finds a good nutrient medium in the meconium.

Czeray attaches much significance to enteral infections in the newly born. According to his experiences at Epstein's clinic it is very noticeable sometimes on the first day. "Even when the children have received no nourishment whatever, they may fall sick with fever and acute symptoms in the gastro-intestinal canal. They vomit the mucous contents of the stomach, the meconium is rapidly evacuated, and diarrhoea sets in immediately. The faeces consist of more or less decomposed intestinal secretion. Sometimes the putrid smell of the faeces, without further examination, proves that there are pathological conditions in the intestine. . . . In a large number of cases feeding with mother's milk is prejudicial to the child, just as in severe alimentary intoxication in older infants. It is necessary to defer the mother's milk for a few days in order to master the processes of decomposition in the intestine. . . . The number of deaths in these particular cases increases terribly, if the mother's milk is not promptly replaced by water and tea diet. The unfavourable alimentary influence of mother's milk admits of hardly any other explanation except that there is a preponderance of bacterial flora in the intestine for which the mother's milk gives a favourable nutrient medium. Under these conditions

the processes of decomposition of the constituents of the mother's milk must be essentially different from the physiological.

The enteral infections, in which the symptoms of disease are noticeable, even on the first day, present a very bad prognosis. For with considerable loss of weight collapse occurs in a few days and can seldom be prevented.¹⁶

In spite of abundant material for observation, in the course of ten years the author was never able to observe these severe general diseases with marked gastrointestinal symptoms on the first day of life, as described by Czerny. Obviously the environment in which the observations were made was different, and the rare occurrence of an earlier and obviously fairly typical clinical picture shows the beneficial effect of better hygienic conditions in modern hospitals.

Gastro-enteritis is possibly partly acquired intra-partum or even intra-uterine. In the latter case the infection occurs from the swallowing of infected liquor amni—according to Hellendall's researches, a relatively rare occurrence. Baron reports three cases of vomiting and diarrhoea associated with foul-smelling liquor amni at birth. Kröning examined the infant of a mother in a high state of fever during birth, and which died after eighteen hours. In the much distended gastro-intestinal canal he found numerous gas-forming bacilli. In this case the liquor amni had also been infected.

As a rule, it more frequently occurs that decidedly bad stools are evacuated only after a few days, about the third or fourth day of life. There is no doubt that there is an abnormal bacterial process of decomposition of the intestinal contents; the stools are highly offensive, generally dark brown, rather thin and sometimes containing gas. Stools containing blood are sometimes evacuated; it may be considered as proved that many cases of intestinal hemorrhages presenting the appearance of melæna arise from enteral infections. Stools with and without blood may alternate (Kowalewski and Moss). In earlier times severe septic gastro-enteritis with violent vomiting was not uncommon among the newly born. In mild forms the processes in the intestine are often not without influence on the general state of health; even though, in such cases, no sort of severe septic symptoms occur, the children frequently look ill, are restless and sometimes feverish. The varying virulence of the causal agents may produce different clinical symptoms and a different course, at different places and at different times.

"Even the circumstances," Czerny and Keller write, "under which the infection ensues during or shortly after birth, make one suspect that many kinds of pathogenic micro-organisms participate in the enteral infections of the newly born. The numerous varieties of the clinical picture indicate this probability. Even if the effect of pathogenic micro-organisms is only extended to the intestinal contents different clinical pictures will result. Sometimes putrefaction preponderates, sometimes formation of gas in the intestine,

in one case the course is acute and in another its course is protracted. But the greatest diversity is shown when, in severe cases the pathogenic micro-organisms are not confined to the intestine but penetrate the blood and lymph channels. According to the kind of bacteria which participate, symptoms of septicæmia or pyæmia result. It is hardly possible to describe all the aspects of the disease which may be caused by general infection of the intestine in the newly born."

It is difficult to make any general rules for *treatment* of these clinically uncharacteristic forms of disease. If the quality of the stools points to processes of putrefaction of the intestinal contents it is to be recommended that the lower portions of intestine be repeatedly and thoroughly washed out. In the same manner charcoal may be administered on account of its action on bacteria and toxins. The prescription of calomel (gr.005 three times) is thoroughly justified. In severe cases it is wise to order an absolute water diet (tea with saccharine, no sugar) for a short time, half, to one day; feeding with small amounts of mother's milk may then be substituted, but these must for some time be below the level of the normal milk, and indifferent fluids should make up the bulk. Theoretically it would be conceivable, that with severe enteritis some sort of artificial nutrient mixture, such as albumen milk, would be therapeutically superior to mother's milk. But one should be very guarded with such assertions until they have been clinically proved. In those cases in which brown, frothy, mucous stools are evacuated during the transitional period, without threatening general symptoms occurring, suckling should be best continued, as in the harmless irritative catarrhs to which the afore-mentioned disturbances of digestion lead pathogenetically. The theoretical explanation of this would be that the fermentative forms of the gas-phlegmon bacillus, with an abundant milk supply, would be forced into the background through the normal flora of mother's milk. As in the treatment of older infants with disturbances of nutrition, careful individualization is necessary, coupled with accurate observation of intestinal and general symptoms.

If an artificially fed child shows symptoms of severe intestinal irritation, the prognosis is, under any circumstances, far more ominous than with a breast-fed child. Of course, a temporary irritative catarrh may be present, but often a "stadium dyspepticum" exists, leading to "disturbance of balance" or "decomposition." With the small infant, the very difficult treatment is essentially the same as with the older infant and need not be further discussed here. If in an artificially fed child marked signs of intestinal catarrh or incipient disturbances of nutrition are diagnosed during the first weeks, the wisest course is, instead of experimenting, to introduce some form or other of feeding with mother's milk.

Constipation.

Infrequency of evacuation of the bowels or temporary constipation during the first few days, is very often a result of an insufficient amount of food and may be regarded as a sure symptom of under-feeding. That form of constipation which is found in older breast-fed children, particularly in very healthy ones that assimilate food very well, is not usually found in the first week of life. But it is often noticed, that either during the meconium period or after evacuation of the meconium, for twenty-four hours or even longer—no stool is evacuated.

If no evacuation of the bowels continues, it may often be easily accomplished by an injection, an enema (glyc. + water, āā 5.0) a small suppository (e.g., bile suppositories for children) and sometimes even by the mere introduction of an intestinal tube. Internal methods for treatment of constipation should not be used during the first few days. Dietetic treatment should also hardly come under consideration, so long as a child is "newborn." If there is no evacuation of the bowels after the above methods of treatment, it must be regarded as a serious symptom, pointing to the presence of some obstruction of the bowel.

Concretions of Mucus in the Lower Large Intestine and Rectum.

Occasionally a gelatinous mucous plug is seated, like a crest, on the first mass of evacuated meconium, sometimes greyish-white, sometimes light yellow and sometimes somewhat bile-stained in colour. It either changes immediately into the colour of the meconium, or remains as a greyish-white, gelatinous mass on the meconium, with the appearance of a coat of mucus (Merdner). The size of this meconium plug is very variable. According to Cramer, who first described it, it generally weighs from 1 to 2 gr.; its length is 2 to 3 cm., but it may be much longer, up to 8 cm. It consists of masses of mucus, to which roundish bodies are attached, and which plainly owe their existence to cells, from their colour with nuclear staining substances. They contain partly nucleated and partly non-nucleated epithelial cells. Cramer believes that the plug originates from the intestinal secretion which is formed in the lower part of the large intestine and rectum, or, in a portion of intestine in which there is frequently no meconium. The plug is only observed when the first spontaneous evacuation of the meconium occurs. If it is often not seen, the reason is that a large proportion of children have evacuated a slight amount of meconium before birth. The plug may then be found in the foetal membranes. In other children the meconium is found 3 to 4 cm. above the anus, so that the mucous content of the rectum does not belong to the typical plug. It is very difficult to find in the first meconium napkin, as it is often blended with the meconium. This may possibly explain why the plug can so comparatively seldom be found (e.g., by Weil only once in 500 births).

There appear to be larger structures connected with the physiological meconium plug, and are described by many as a pathological condition (Longuet, Ullmann, Berti, Trumpp). They are mucous concretions, whose structure and chemical quality vary considerably in individual cases. They have a rounded sausage-shaped form, are whitish in colour, of flabby or dense consistency, contain small ball-like lumps of mucus, epithelial cells, occasionally fibrin ("fibrinous-epithelial concretions."—Berti), and sometimes lime concretions (Trumpp). It appears that such structures sometimes cause symptoms of temporary intestinal obstruction; as a rule they are evacuated spontaneously before or together with the meconium.

Trumpp attributes the existence of these epithelial mucous plugs to foetal enteritis membranacea, such as Rochmann has described in a newborn child: A child with symptoms of ileus, from whose intestines only one large ball of meconium covered with mucus, and later more mucous pseudo-membranes were evacuated by means of enemata, died on the twelfth day after formation of an anus *præternaturalis*. The post-mortem showed volvulus of the intestine in the lower part of the ileum, and in the colon masses of pseudo-membranes attached to the intestinal wall. The glands of the mucous membrane of the large intestine were filled with mucus, which continued as far as the lumen of intestine.

Occasionally in the meconium is found a collection of soft mucous bodies the size of a pea of the same colour as the rest of the faeces.

They are probably genetically connected with mucous plugs. No pathological importance is attached to them.

Congenital tumours of the gastro-intestinal canal are pathological rarities and therefore of no essential clinical interest. Leube-Wilkinson and Widerhofer have described cases of congenital cancer of stomach; Cullingworth, a carcinoma of the pylorus causing stenosis in a child of five weeks; Ahlfeld a carcinoma of the rectum in a stillborn child.

Meckel's Diverticulum.

Meckel's diverticulum is the expression of a persistence of the ductus omphalomesentericus, the passage leading from the bowel to the vitelline sac, which, under normal circumstances, closes in the eighth week of foetal life. The diverticulum is an intestinal appendix at the lowest part of the ileum, variable in length and diameter.

Roth gives the following description of the various forms and positions of the diverticulum:—

- (1) The common form of Meckel's diverticulum is situated:
 - (a) in the abdominal cavity, wherein it terminates either as a ligamentous process, or more as a cord floating free;
 - (b) more rarely in a hernial sac;
 - (c) still more rarely within the mesentery.

(2) The adherent diverticulum: by means of its blind end or a cord the outgrowth is generally attached at the navel, more rarely at another part of the abdominal cavity.

(3) The open diverticulum: it discharges into the navel:

(a) open diverticulum in a more limited sense;

(b) overtopped by a small prolapse of wall;

(c) overtopped by a red hollow appendage;

(d) complicated by a secondary prolapse of intestine (fig. 48).

(4) The diverticulum is the place or origin of retention cysts, in which:

(a) communication with the intestine is retained; or,

(b) interrupted.



FIG. 48.—Meckel's diverticulum with prolapse of the intestine.

In the last case the formation of a sub-peritoneal or subcutaneous cyst may result.

The open diverticulum is of chief interest in the clinical pathology of the newly born. After the separation of the stump of the umbilical cord, in the region of the umbilical wound a fistula occurs from which a thick secretion is discharged. The fistula is generally very small, the secretion not very great; on superficial observation therefore the lesion may easily be overlooked. More rarely, the opening of the fistula is wide and the

secretion considerable. The opening in most cases consists of a small orifice in the centre of the prolapsed mucous membrane. The affection may be easily confused with an umbilical granuloma, though the surface of the latter is hardly so smooth as that of intestinal mucous membrane. If the presence of the mouth of a fistula has been established, it is possible to mistake it for a urachal fistula. As a means of differentiation the reaction of the secretion should be tested, which is usually alkaline in an intestinal fistula and acid in a urachal fistula. The secretion should be examined microscopically.

The best treatment of Meckel's diverticulum is operation: extirpation of the outgrowth as far as the small intestine through a small incision along the median margin of the rectus muscle, invagination of the stump and suture of the peritoneum over it (Spatz). The operation need not necessarily be undertaken during the first few weeks, as generally there is no immediate danger. The latter only occurs if, owing to continual prolapse of the outgrowth, the mesentery which is adherent to it is caught by the umbilical ring; symptoms of ileus may then arise (Kerih, Seitz). The conservative method of treatment (cauterization, or use of silver nitrate) may sometimes close the fistula but is not to be recommended.

Stenosis and Atresia in the Region of the Digestive Tract

(1) OESOPHAGUS.

Over one hundred cases of atresia of the oesophagus have hitherto been published (Kreuter, Diehle, Vieillard and Le Mée, Wunsch, Latener, &c.). In the overwhelming majority of cases there exists a communication with the trachea besides the occlusion of the oesophagus. The upper section of the latter forms with the pharynx a cul de sac, the end of which lies at the cricoid cartilage or lower down, corresponding to the seventh or eighth tracheal ring, or still lower, say at the bifurcation of the trachea. To this cul de sac a more or less distinct cord-like central portion is attached, leading down to the lower part of the oesophagus which is connected with the trachea. The mouth of the communication may be small, fissure-like, or wide; the lower part of the oesophagus then forms, as it were, the continuation of the trachea. In rarer cases communication with the trachea is absent, there is instead a more or less long and solid central portion situated in the course of the oesophagus. Occasionally the obliteration is so extensive that the oesophagus appears to be altogether lacking; at other times there is only a thin membrane-like septum (Marsh). Most rare is a connection of both oesophageal segments with the trachea.

Atresia of the oesophagus is generally found in otherwise well-developed children whose external appearance immediately after birth presents nothing to attract attention. The characteristic symptoms only appear with the first efforts at taking nourishment. Swallowing

is impeded; after a few successive rapid swallowing movements the child emits gurgling sounds, "swallows the wrong way"; the fluid in the cul de sac gets into the trachea. Violent attacks of coughing and vomiting occur with threatening asphyxia. With each effort to swallow the suffocatory attacks are repeated, according to the capacity of the cul de sac immediately after the first or after several movements of swallowing. As a rule the children suck voraciously. As long as meconium is present in the intestinal canal stools are passed by the child, exhibiting nothing remarkable in this respect. As soon as the meconium is evacuated, either complete constipation follows, or at the most, scanty stools consisting of intestinal secretion. As a rule these children die from inanition between their third and seventh day, but exceptionally not until the second week (from inanition) and unavoidable pulmonary complications in consequence of aspiration of milk.

From a differential diagnostic point of view it is important to note that a similar symptom-complex may be found with cleft palate; therefore a very thorough inspection of the mouth must not be omitted. There also may exist an oesophago-tracheal fistula with an otherwise normal oesophagus and trachea; with the assistance of a stomach tube the diagnosis can be immediately established.

Treatment is utterly useless, especially in the case of the relatively frequent communication between the oesophagus and trachea. The best surgical treatment is gastrostomy, or if the atresia is situated very high up oesophagotomy might be considered. Hacker suggests, after the establishment of a gastric fistula, connecting the two parts of the oesophagus by means of a Murphy's button. But even if a child should survive such an operation it would be very liable to succumb to aspiration pneumonia. Operative treatment should be therefore avoided.

Stenoses of the oesophagus are uncommon. They need not necessarily prove fatal. Up to the present no examples have been found in autopsies of newly born (Kreuter).

(2) STOMACH.

Congenital obstructions observed up to the present occur exclusively in the pylorus.

(a) *Atresia of the Pylorus.*

This is a very rare occurrence. Kreuter reports on only four cases collected from literature. The author observed an atresia of the pylorus in a child of an eclamptic mother that died on its first day; the remainder of the alimentary tract was in this case normal. The symptoms of occlusion of the pylorus are similar to those of the much more frequent duodenal atresia.

(b) Congenital Stenosis of the Pylorus.

Of the three different types of stenosis occurring in the infant, two must undoubtedly be considered as congenital:—

(a) The congenital narrowness of the lumen of the pylorus, the so-called Landerra-Meyer type of stenosis. It constitutes a congenital anomaly of development, the mucous membrane of the pylorus being remarkably thin in its developed state. In contrast with this is the far more frequent hypertrophic form of pyloric stenosis, in which folds of mucous membrane invariably form, and the mucous membrane has a normal thickness (Wernstedt). From a clinical standpoint this extremely rare anomaly has no essential interest in the pathology of the newborn. It appears to run its course without symptoms.

The first case of this kind in an infant was recently described by Schäfer, in which the malformation consisted of a small fistula-like pylorus, without any sphincter muscle. In spite of the narrowness of the lumen of the pylorus the child lived for ten months. The stenosis led to secondary dilatation and hypertrophy of the stomach, and also to hour-glass contraction.

(b) Anomalies of formation and connective tissue stenoses which aetiologicaly correspond to the congenital occlusions in the remainder of the digestive tract. This includes either simple septa of mucous membrane or adhesions and cords, which may originate from foetal peritonitis (Mya). It naturally depends on the degree of the contraction, whether and how long a child afflicted with this stenosis survives.

Both of the above mentioned types are extremely rare. The most common type of pyloric stenosis and relatively the most frequent cause of obstruction in the digestive tract, occurring during infancy, is the so-called Hirschsprung type of stenosis, the hypertrophic form of pyloric stenosis and pylorospasm.

The aetiology and course of these forms of disease have been most carefully studied in recent years (Plaundler, Wernstedt, Ibsahim). The typical post-mortem finding consists in a hypertrophy of the musculature of the pylorus, and it is probable that the cases of pylorospasm which recover also exhibit this hypertrophy of the pylorus. It is another question whether the latter is to be regarded as congenital or as a secondary acquired condition due to a functional hypertrophy from spastic contractions. In the first case it might be a malformation of the pylorus or a functional hypertrophy, but have existed as an intra-uterine condition (Thomson). It is difficult to decide the nature of the irritants that lead to these congenital hyperplasias. They might consist of swallowed liquor amnii and irritant matters therein present, nervous disorders, irritants from the intestine, &c. (Alder). But such considerations concern only those relatively rare cases of pylorospasm, in which the characteristic symptoms already exist at birth or in the first days of life. They can only be found in a minority of cases.

The disease generally begins at the end of the second or third week, frequently still later.

The following are the statistics of Ibrahim (1906) concerning the time of onset of vomiting, the cardinal symptom of pylorospasm:—

| Onset of Vomiting | No. of Cases |
|-----------------------|--------------|
| First-third day ... | 51 |
| End of first week ... | 24 |
| " " second week ... | 53 |
| " " third week ... | 64 |
| " " fourth week ... | 34 |
| " " fifth week ... | 11 |
| " " sixth week ... | 14 |
| " " seventh week ... | 3 |
| " " eighth week ... | 5 |

The fully developed clinical picture of pylorospasm is as follows:—

(1) Vomiting, either after every meal or—with very small quantities of fluid—only after several meals. The amount of vomit occasionally exceeds that of the last meal (stagnation). The vomiting is projectile.

(2) Constipation: complete absence of stools or scanty lumpy stools.

(3) Distension of the epigastrium, sunken hypogastric region, visible gastric peristalsis, occasionally obvious pain due to colic, sometimes a palpable pyloric tumour.

This typical clinical picture is practically never seen in the newly born. If the above symptoms are noticed, it is far more probable that an atresia or stenosis in the upper small intestine is present. The factors producing pylorus cramp, which by the majority of authorities is considered an essential factor in the condition of hypertrophic pylorus stenosis, may during the first days of the infant's life lie under the stimulus threshold, owing to the small quantities of fluid taken. The disease, therefore, at first remains latent, even if the vomiting manifests a persistent character. During the first few days vomiting is a very frequent symptom, and it would be seldom possible to recognize, in a solitary case, its pylorospastic nature. Pylorospasm is a disease of infancy, the first signs of which may date back to the first days of life, but the fully developed symptom-complex may only be attained at the end of that period classified as newly born.

It is very difficult to decide at the outset of the disease soon after birth, whether pyloric stenosis is present, either functional or spastic (without anatomical changes) or true congenital hypertrophy. There are no convincing post-mortem results proving the presence of a hypertrophic pylorus in the newborn. No conclusions can be drawn from the few cases collected from literature (Ashby, Simonsohn, Demé, Delamare and Dienlaffé). Ibrahim believes that the disposition to a spastic condition is congenital and perhaps based on an abnormally strong musculature of the antrum pyloricum.

The morbid conditions of the pylorus in the newborn responsible for hypertrophic stenosis or pylorospasm are as follows:—

(1) Hypertrophy or hyperplasia of the musculature;

(a) Malformation; anomaly of development.

(b) Functional hypertrophy of intra-uterine origin.

These changes in the pylorus may produce symptoms of stenosis or form the basis of a disposition to spasms which become manifest during the first few days of life or later.

(2) Functional stenosis with an anatomically unchanged pylorus. Spasms of pyloric musculature of nervous origin produced by food irritants, &c., which only lead secondarily to hypertrophy.

We will not go further here into the treatment of pylorospasm. As far as the newborn child is concerned it is the same as in the case of vomiting.

Stenosis in the region of the pylorus may also occur from compression. Toporski reports a case of pylorus compression by the caecum, associated with displacement of the intestine.

(3) SMALL INTESTINE.

Among congenital obstructions of the digestive canal those of the small intestine are the most frequent and clinically the most important. There are about 200 reports of cases in the literature in about a third of which the duodenum is concerned, which bowel therefore seems to be a favourite site. Atresias are relatively much more frequent than stenosis. The clinical picture of the latter does not deviate essentially from that of complete intestinal obstruction. Most commonly there is a membrane or a folding in of the mucosa leaving a small central opening, which does not, as in the oesophagus which forms a rigid tube, remain permeable, but very soon becomes occluded (Kreuter, Preisich).

The duodenal atresias are most frequently localized in the region of the papilla duodeni and the duodeno-jejunal flexure; but they are also found in other parts of the duodenum. The proximal and distal portions of the intestine are situated either in close apposition or are connected by a cord, which may however be absent, so that there is complete separation of the two parts of the bowel. The lengths of the impervious portion may vary in extent. In extreme cases it may reach from the pylorus to the duodeno-jejunal flexure (Albers).

Of diagnostic importance is the condition of the common bile duct. In the majority of cases it opens into the caudal section, less frequently above the atresia (Weber). Occasionally there is a division of the common bile duct, each passage opening into the upper and lower portion of intestine (Hauser, Karga). Kermanner interprets these cases as stenoses and considers the supposed second duct to be the lumen of the intestine.

In the jejunum and ileum there are often multiple obstructions, just as stenoses and atresias in the upper part of the gastro-intestinal

canal are found in various places. Stenoses and atresias may also exist in close proximity to one another (Kuliga). Here also any intestinal segment may be affected, a favourite site is the lower ileum in the vicinity of the caecum. As in the duodenum, the impervious parts in the remainder of the small intestine vary in length and all varieties may be found, from the merely membranous septa to complete interruption of continuity. The intestinal segment situated above the atresia is generally greatly dilated, and its walls hypertrophic, the caudal segments of intestine are, as a rule, contracted, and not infrequently extremely hypoplastic.

The clinical picture of intestinal obstruction is only fully developed when the child begins to take nourishment. But even on the first day of life the condition of the meconium may give certain clues and arouse suspicion of the existence of an intestinal stricture. Normal coloured meconium will only be evacuated when the stenoses or the atresia are situated above the opening of the bile duct. At this period also the absence of kugugo in the meconium may be of diagnostic importance as it points to the fact that none of the swallowed liquor amnii has reached the lower portion of the intestine (Walz). If the obstruction is situated below the papilla duodeni or in a lower portion of the small intestine, no meconium whatever will appear. After an enema, the contents of the intestine are evacuated, but the latter merely consist either of light grey or greenish, viscid mucus, or of thickened crumbling chalk-like masses. The amount depends on the localization of the obstruction.

Apart from these disquieting symptoms, at first the child manifests nothing out of the common, it frequently looks quite well, and sucks vigorously when first put to the breast. But the characteristic symptoms soon appear. Sooner or later, according to the position of the obstruction, the child vomits after each meal; the vomit is generally bile-coloured, with a lower seated obstruction it gradually acquires the appearance of intestinal contents; admixtures of blood also appear occasionally. If one watches the abdomen, after a feed, one can see plainly visible the intestinal loops standing out through the abdominal walls. In the course of a few days considerable meteorism may develop. The abdomen is then generally tender on pressure. The visible peristalsis of the distended intestinal loops presents a very striking appearance. All spontaneous evacuation of the bowels ceases. The secretion of urine is minimal, occasionally completely suppressed. Although the child sucks, sometimes even voraciously, it loses weight rapidly. Breuschneider considers that a marked decrease of weight (several hundred gm.) up to the second day is of diagnostic importance.

The diagnosis of atresia is only justified if, besides vomiting and peristalsis, the absence of coloured intestinal contents is established, and especially of stools containing remnants of milk. As already mentioned, the same aspect exists in stenosis due to occlusion of the usually small opening. If exceptionally the bowel

remains patent, the intestinal contents may be evacuated; but the motions are, as a rule, scanty, occurring at intervals of several days and generally only with enemas. Stenoses of the small intestine may occasionally be more easily passed by the liquid intestinal contents than lower seated stenoses; scanty evacuation of bowels may occur daily (Trumpf).

Under certain circumstances the visible peristalsis may afford information as to the localization of the point of obstruction. As with pyloric stenosis the peristalsis chiefly affects the stomach; so with duodenal atresias and stenoses, and it is correspondingly visible in the epigastrium. The form of the distended portion is sometimes like that of hour-glass contraction. The constriction corresponds with the pylorus and the distended portions with the stomach and distended upper duodenum. With a lower situation of the stenosis the peristalsis is generally visible throughout the abdomen. It is hardly possible to decide with exactitude whether it is a case of obstruction in the small or large intestine. When the stenosis is high up the meteorism may be confined to the epigastrium, whilst the hypogastric region remains soft. It goes without saying that in all cases an examination must be made of the rectum to eliminate the possibility of imperforate anus or rectum. Trumpf points out that sounding the intestine with an instrument may be deceptive, as an obstruction of the passage of the instrument may be produced by a crooked and collapsed or spastic sigmoid colon.

The prognosis is quite hopeless. The children usually die within the first week, more rarely in the second, and very exceptionally in the third week, from inanition, aspiration pneumonia, peritonitis or intestinal rupture. A longer survival has been observed with stenosis (Theremin twenty-six days, Denne four months, Grunberg seven weeks). A unique case was that of a child, observed by Finkelstein, that lived twenty-eight days with ileo-cæcal atresia.

The treatment can only be operative—but the results have not been promising—up to the present no child operated on has recovered. This may partly be due to the fact that the diagnosis has been established too late and the child operated on in too bad a condition. What, *a priori*, destroys the prospect of success with an operation is the relatively frequent multiplicity of strictures, as well as the rudimentary development of the portions of the intestine below the stricture. Even if an operation is resorted to with but the faintest hopes, it at least affords the only possible expedient. Gastro-enterostomy with duodenal strictures and entero-anastomoses with strictures of the small intestine are the only correct operations. The formation of an artificial anus frees the child from the distressing symptoms of meteorism and accumulation of feces, but only with a low position of the stricture can life be retained or prolonged. Trumpf therefore always recommends gastro-enterostomy. He is of the opinion that in the absence of multiple stenosis the possibility exists that the food in the stomach if it be passed

through the larger portion of the intestine may be of use. v. Tschendloff succeeded in feeding a child, that had undergone enterostomy six days after birth, for fifteen days through the intestinal fistula. Braun suggests opening the abdomen in the median line on the second or third day, taking out the proximal and distal portions of intestine and anastomosing both together as quickly as possible by means of clamps.

(4) COLON.

Atresias and stenoses in the region of the colon are far more rare than those of the small intestine. According to Krüger's statistics, in 50 per cent. of the cases there are also obstructions of the lumen of the small intestine. In the large intestine itself there are sometimes multiple occlusions and stenoses (Sperlas). In pure cases the symptoms resemble those of stenosis of the small intestine. In considering the low situation of the obstruction it may be expected that vomiting will start later and retention of faeces sooner. If, apart from mucus, there is no evacuation of the bowels, it points to a low situation of the occlusion, though this is also possible with an atresia situated high up, if its formation dates back to the first five fetal months. With stenoses of the colon evacuations occur periodically with sufficiently increased pressure and generally with pain. Trumpp observed in a case, in which the stenosis was situated above the sigmoid (due to axial rotation of the mobile part of the colon following partial adhesion of the persisting meso-colon to the parietal peritoneum), periodical vomiting, which subsided when the bowels were evacuated at rare intervals.

Treatment, from a theoretical standpoint, is the formation of an artificial anus or entero-anastomosis (ileo-colostomy). But hitherto neither of these operations has succeeded.

The origin of congenital stenoses and atresias in the region of the digestive tract has not yet been fully explained. Possibly the same explanation may not meet all cases. The following factors are suggested as partly explanatory: (1) Mechanical factors; (2) inflammatory processes; (3) embryonal disturbances of development.

(1) In isolated cases an enlarged liver, a hypertrophic head of the pancreas, congenital abdominal tumours or cysts may compress externally a part of the intestine and give rise to symptoms of stenosis. Such causes cannot, however, as a rule, be found with complete strictures.

As in later life, a volvulus and an intussusception may lead to intestinal obstruction *in utero*. Such a fetal axial rotation of the intestine may have various causes: extension of the umbilical cord torsion to the intestine (Tillmanns), twisting of the intestine round its longitudinal axis, owing to anomalies of the mesenteric attachment, or owing to differences in growth between the mesentery

and the development of the intestine, fixation of the bowel through the ductus omphalomesentericus or Meckel's diverticulum, or through peritoneal bands. The occurrence of a fetal volvulus (Schottelius, Garner, Späthel, Leichtenstern, Huttenbrenner) is disputed by Kuliga and Kreuter; Hoffmann also does not consider torsions of the intestine to be the cause of atresias, but as terminal phenomena. According to Trumm, the circumstances in favour of the aetiological explanation of a volvulus from umbilical cord torsion are that while the intestines are occupying the umbilical cord the duodenum and caecal segment are situated at the level of the later umbilical ring, and therefore with the supposed twisting of the umbilical cord are subjected to severe pressure; these are the parts of the intestine in which most stenoses and atresias have been observed. Chiari and Braun both found intussusceptions. Kreuter considers the latter, not as causes, but as results of stenoses. In one case (Fuchs) there was incarceration of the small intestine in an abnormal cavity of the mesentery, a kind of retroperitoneal hernia.

(2) Fetal inflammation, which is proffered as explanation by Thoremin, Fiedler, Simpson, Tobnitz, &c., may affect either the peritoneum or the intestinal mucous membrane.

According to experiences in adults, it seems quite plausible that peritonitis from adhesions, cord formations and kinkings can be sufficient cause for the obstructions in the wall of the gut, though in the majority of cases no remains were found of fetal peritonitis. And it is not quite comprehensible why pre-natal inflammation should specially attack the duodenum and lower ileum (Kreuter). One inclines more to the opinion that peritonitis is rather a result than a cause of the atresia. Even if in isolated cases a peritoneal band may cause a stenosis, such a condition is by no means frequent.

Fetal enteritis also, which was supposed to exist, owing to the various anatomical changes, such as over-filled vessels, swelling of the intestinal follicles, and the presence of positive bacteriological findings, should be more reasonably regarded as a secondary process.

(3) With regard to those hypotheses which are based on the acceptance of embryonal anomalies, those in connection with Meckel's diverticulum and ductus omphalomesentericus have already been mentioned. In other cases vessel anomalies with partial insufficient nourishment of the intestine (Wyss), as well as occlusions through hypertrophic folds of the mucous membrane, have been assumed (Hammer, Schnitzlein, Wyss). Oberndorfer attributes the pathological formation of folds to accessory glandular germinations derived from a rudimentary accessory pancreas.

Even if these aforementioned causes may apply in many cases, probably no general validity for any can be claimed. There are most adherents to the hypothesis which attributes stenoses and atresias to the arrest of an early phase of the development, and

regards them as pure malformations (Tandler, Kreuter, Kersten). In the first weeks of fetal development atresias occur in the fore gut, mid gut, and hind gut, temporary obliterations which represent a normal process of development of the early embryonal entoderm tube. They arise through cell proliferation in the already fully developed intestinal tube, and from which results a solid condition of the gut in various portions of the intestine. If the embryonic formation does not disappear from some unknown cause an atresia remains; if it only partially disappears, a stenosis remains. The entoderm is destroyed and subsequently replaced by connective tissue. With regard to duodenal stenosis, Meusburger is of the opinion that within the quickly growing region between the pylorus and origin of hepatic outgrowth (intercalary duodenal region of Benecke) an abnormal arrest of growth of the entodermal tube may lead to an atresia. According to Kermanner, the real cause is not to be found in a disorder in the region of the entoderm, but in that of development of the mesoderm, the muscular wall; in his opinion the causal origin might be found in chemical toxic factors. Occasionally hereditary influences appear to play a part.

(5) RECTUM AND ANUS.

The following malformations occur in the region of the hind gut:—

(1) *Atresia ani*.—The anal opening is closed by a membrane. The skin is either drawn right over the anus, or in its place there is a shallow dimple or small fold above which the hind gut terminates in a kind of cul de sac.

(2) *Atresia recti*.—The rectum is partially or completely replaced by a solid cord. The sigmoid flexure terminates at the sacrum or still higher up by an impulla. In mild cases the atresia may be confined to a short band of tissue. With the pure form of atresia recti an external anus is present; but above the latter an obstruction is found to the finger. If there is simultaneously an atresia ani, the term used is atresia ano-rectalis.

(3) *Abnormal openings and communications*.—The rectum does not terminate at the normal place, but in the region of the urinary passages or the genitals.

Atresia ani vesicalis, opening in the bladder. If the opening of the fistula is sufficiently wide, the malformation does not necessarily lead to obstructive symptoms, but is very dangerous owing to the invariable occurrence of infection of the urinary passages.

Atresia ani urethralis, opening into the urethra, at the under surface of the penis near the scrotum.

Atresia ani vaginalis, opening into the vagina. In this case the prognosis is not so unfavourable as in the aforementioned forms, if there is a sufficiently wide communication.

Atresia ani vestibularis, opening in the vestibulum vaginae. As

in this case the rectum opens externally, the prognosis is relatively good, provided the openings are sufficiently large.

In all the above malformations, in addition to the abnormal communications, there may be a normal orifice of the rectum. These malformations would then constitute fistulae.

The symptoms of complete atresia of the rectum, as in those cases in which an opening is present, though the lumen is very narrow, are those of intestinal obstruction; complete constipation, after a time, vomiting, meteorism, visible peristalsis, &c. With a little observation it is easy to form a diagnosis. In a normal anal opening it is easy to palpate the blind end by digital examination of the rectum; diagnostic difficulties will only occur if the atresia is very high up. Examination with a bougie is deceptive. Abnormal communications and orifices may be recognized by the appearance of faeces in the urethra or vagina, or in some part of the peritoneum, but it must be taken into consideration that admixture of faeces with contents of the bladder cannot be found in every portion of urine.

The earliest possible recognition of atresias in the region of the hind gut is of the utmost importance, as in the majority of cases an immediate operation is the only means of saving the child. If the sequelae of intestinal obstruction occur, the prognosis is much worse. It is only permissible to wait with atresia vesicularis and, under certain circumstances, with atresia vaginalis.

The operations, which need not now be further described, aim at allowing not only the free passage of faeces, but also at restoration of a normal and proper functioning rectal orifice. It is most practical to seek for the blind end of the rectum from the perineum. A blunt perforation is only to be recommended if the interposed layer is so thin that the meconium can be seen through. If this layer is thicker, after a median incision, the rectum must be sought for on the front surface of the sacrum, and if possible the unopened ampulla fixed to the skin, and only opened then, or, if this is not possible, the mucous membrane of the opened ampulla should be stitched to the external skin (proctoplasty). If the proctodaeum cannot be found, it must be sought by laparotomy from the abdominal wound and passed downwards to the perineal wound. The formation of an artificial anus should be the last resource (Spuy).

Hirschsprung's Disease and Allied Conditions.

Although the so-called Hirschsprung's disease does not correspond to any aetiological uniform conception, it represents a fairly circumscribed group of diseases. Its characteristic peculiarity lies in the abnormal width and thickening of the walls of the large intestine (megacolon), with absence of constrictions and obstructions in the lumen of the dissected intestine. The cases of Hirschsprung's disease are thereby quite distinct from those with an anatomically established stenosis. There is no uniform answer to the question

as to how dilatation and hypertrophy of the colon (megacolon congenitum) occur, and it appears as if several of the hypotheses brought forward might be right (Kleinschmidt).

Of the various factors concerned in the aetiology of Hirschsprung's disease, the most important is the kinking of the intestine associated with congenital narrowings and twisting of parts of the large intestine, in particular of the sigmoid flexure. The infantile flexure exhibits a relatively greater length than that of the adult. The proportion of the length of the large intestine to that of the body is $2\frac{1}{2} : 1$ (against $2 : 1$ with adults), whereas the ascending and transverse colons are relatively short, the descending colon and, in particular, the sigmoid flexure, longer. The relatively greater length and movability of the intestine, and therefore the opportunity of twisting, create a disposition to kinking which may result in an obstruction of the lumen. As explanatory of the pathogenesis of these cases presenting, after birth, the aspect of megacolon, the condition pointed out by Konjetzny is particularly important. In a child of three days he was able, by fixation of the abdominal viscera *in vivo* with injection of the vessels with formalin, to find a kink of the sigmoid colon at its junction with the rectum, which, in intra-uterine life, had led to stasis of the meconium in the transverse and descending colon, also in the abnormally large, much twisted sigmoid, and to dilatation and hypertrophy of the walls of these portions of intestine. The longer and more movable the sigmoid flexure, the more easily can the kinking occur. The kink is either situated at the junction between the sigmoid and the rectum, or at the junction between the descending colon and sigmoid colon. According to Pfisterer, the length of the loop is not so important as that of its mesentery, and with sufficient movability a kinking of the sigmoid is also possible without elongation.

Apart from the kinking a volvulus of the loop can also lead to an obstruction of the lumen. This factor is of less importance during the period of infancy, as the fixed ends of the sigmoid flexure are not close to each other, as is necessary with a volvulus, but are far apart (Heller). Only with the requisite close proximity of these non-mesenteric ends, a rare anomaly, a volvulus may occur with young children (Kleinschmidt).

In other cases it appears that a spastic contraction of the intestine may lead to development of Hirschsprung's disease even in very young children (Koeppel). If the obstruction is within reach of the finger, it is possible, with digital examination from the rectum, to palpate a narrow ring corresponding to the spastically contracted portion of intestine. If the spasm has not led to hypertrophy, the real cause of the trouble will probably not be discovered even at the autopsy. In such cases dilatation, stoppage of the faeces and kinking may occur secondarily. (See cases mentioned below.)

The symptoms of Hirschsprung's disease with paralysis of the

rectum and pelvic floor have not yet been observed in the newly born (Finkelstein). The same applies to a valve mechanism due to abnormal development of the plicae transversales of the rectum.

It does not appear as if all cases can be traced to mechanical causes, but that the theory of primary malformation has also a certain justification. In a case of Marchand there was a considerable enlargement of the ascending and transverse colon with gradual reduction of the size in the region of the descending colon, till the relatively narrowest place was reached at the transition to the sigmoid flexure. Besides dilatation and hypertrophy involved by a mechanical obstruction, Kleinschmidt recognizes the existence of true megacolon congenitum idiopathicum in the same sense that Hirschsprung describes it.

The three principal symptoms of fully developed Hirschsprung's disease are the following: great tympanitic distension of the abdomen, severe constipation, visible and palpable peristalsis



FIG. 46.—Meteorism and intestinal distension in Hirschsprung's disease (first week of life).

(fig. 49). Occasionally vomiting and pronounced symptoms of ileus occur. The severe disturbance of the general condition is an effect of the stagnation of the contents of the bowel, of injury to the wall of the intestine, of the abnormal conditions of absorption created thereby, and of insufficient nourishment. The compression of the thorax from the high situation of the diaphragm leads to disturbances of respiration and circulation.

It depends on the degree of anatomical changes in the disease, also on the time when the sequelae appear, whether the disease is manifest soon after birth, or whether it develops only after a more or less long period of latency. The onset is frequently insidious, but the first stages of the disease often date back to the earliest period of infancy, even in the gradually developing forms. According to statistics collected by Löwenstein, out of eighty-eight cases, in fifty-nine the symptoms of disease were present from birth. The children even arrive into the world with distinctly increased abdominal circumference, but more frequently the tympanitic distension appears during the course of the first few days. The second cardinal symptom, constipation, appears during the first few days more frequently than the meteorism. Evacuation of the meconium may fail entirely or only occur after enemata. Even if

the meconium has been passed, constipation is constant. Needless to say, it is very difficult to decide at this period whether the constipation is a result of the disease or of deficient nourishment, for in the bad general condition of the children very little nourishment is taken during the first days. If sufficient milk is taken, vomiting frequently occurs; the vomit may be coloured with bile. If sufficient nourishment or any fluid reaches the intestine, the third cardinal symptom, visible peristalsis, comes into evidence. Well-marked peristaltic waves pass over the distended abdomen. The aspect of the disease has, at this juncture, considerable similarity with an atresia. If a rectal tube is employed one may be not only certain that there is no atresia ani or recti, but that, from the passage of flatus and coloured intestinal contents, there is also no impassable spot in the higher segments of the intestine. The clinical symptoms present are similar to those of a stenosis situated in the lower portions of the intestine.

The immediate consequences of the obstruction of the lumen depend entirely on the degree of the stenosis. If by therapeutic methods sufficient evacuation of the bowels is insured, or if the obstructed contents are passed spontaneously, even at long intervals and if the child takes sufficient nourishment, life may be prolonged. The disease then takes a more chronic course; the children may survive not only the period of infancy but also a few years. If from the beginning the symptoms are severe the children fall into a state of extreme cachexia with all its consequences, especially diminished resistance to disorders of nutrition and infection; or else they succumb with symptoms of inanition. Still, it appears as if the prognosis of Hirschsprung's disease is not invariably unfavourable. In abortive forms, after the first signs of stenosis, the symptoms may permanently disappear with strengthening of the muscular walls of the twisted intestine during the course of growth. But apart from these, in cases of the fully developed symptom-complex, recovery, or, at least, a long period of a permanent state of latency may be achieved under suitable treatment. It is, of course, another matter whether a complete cure may be attained. Heller believes that those children afflicted with a congenitally large and abnormally situated sigmoid flexure, if they do not die in infancy from Hirschsprung's disease, become liable in later life to valvulus of the sigmoid flexure.

Treatment. If there are symptoms leading one to suspect Hirschsprung's disease in the newly born, every effort must be made to remove the obstruction to the lumen. With the usually low situation of the obstruction at the upper end of the rectum, this is best achieved by introduction of a rectal tube. This alone may occasionally alleviate the symptoms for the moment by allowing gas and intestinal secretion to escape. The contents of the bowel must, if possible, be removed daily by means of enemata; it is advisable to inject relatively large quantities (½-litre) as the twisted intestine can thereby be straightened. Very successful results are

occasionally effected by permanent drainage of the constricted spot by means of a soft intestinal tube (Göppert, Zarfl). Nourishment is given according to the usual rules which are employed where there are difficulties in suckling and vomiting. In the rare cases when one is justified in presuming a spasmodic condition of the intestinal musculature, injections of atropin may be tried.

Later on, but in any case not during the period of infancy, an operation may be considered if there are urgent indications; it is not impossible that Hirschsprung's disease may cause symptoms of complete intestinal obstruction—a successful operation under these conditions is very doubtful.

In typical Hirschsprung's disease the obstruction is situated in the sigmoid, the distension of the intestine is in the colon only. Exceptionally, similar conditions appear in other parts of the intestine. In the body of a child of two days that suffered from symptoms of ileus, Torkel found tremendous cylindrical dilatation of the jejunum; which he attributed to "change resulting from disturbance of development." Death occurred from multiple kinking complicated by peritonitis.

A very peculiar case was observed by Reynes: In a child with distended abdomen, meteorism, constipation and bilious vomiting, he found, at an operation undertaken on the third day of life, the ascending colon and the first half of the transverse colon distended and full to bursting, whereas the second half of the transverse colon, the descending colon and the sigmoid flexure were empty and contracted. As a mechanical obstruction was found neither at the operation nor at the post-mortem shortly after, a paralytic condition of the intestine was assumed, a state of hypotonus of the otherwise normal intestinal musculature from deficient innervation (Bing).

The author observed the following case: a premature child weighing 1,700 gm. ($3\frac{1}{2}$ lb.) evacuated meconium on the first day, but then suffered from complete constipation. On introduction of a rectal tube a little bloodstained mucus was passed; water enemata were returned quite clear. Nourishment by means of a stomach tube; frequent vomiting; aspiration pneumonia. Collapse quickly followed. No meteorism, no visible peristalsis. Death on the fifth day. At the post-mortem the ileum and large intestine were found to be contracted and free of contents, apart from a small quantity of greyish stringy mucus. The stomach, duodenum and jejunum were, relatively, well filled, the mucous membrane being in a condition of hemorrhagic inflammation (stagnation).

In another child (weighing at birth 3,400 gm., $7\frac{1}{2}$ lb.) during the first few days there were marked symptoms of intestinal stenosis; distension of the abdomen, visible peristalsis, continual biliary vomiting, absence of spontaneous evacuation of the bowels; only with vigorous massage of the abdomen, relatively large quantities of decomposed meconium could be passed. After an injection of 0.1 mg. atropin on the fifth day (post or propter hoc?) the

symptoms began to disappear completely and the child rapidly recovered. It is not impossible that this was a case of spasm in the lower part of the small intestine.

Such cases prove that in the newly born more or less pronounced symptoms of occlusion or stricture of the intestine can exist without there being a mechanical obstruction in the form of a real atresia or stenosis, a kinking or torsion. The aetiology of these rare diseases is still obscure.

(D) Diseases of the Peritoneum

PERITONITIS.

Both acute and chronic peritonitis may be observed in the newborn child. The chronic form occurs in intra-uterine existence and is therefore known as *foetal peritonitis*.

(1) *Foetal Peritonitis.*

Foetal peritonitis may occur through infective agents or toxic matters being transmitted from the mother to the fetus. Mya believes that such transmission of toxic substances, as in tuberculosis of the mother, may cause adhesive peritonitis. It may be accepted as certain that congenital peritonitis may have a syphilitic basis. In twenty-five cases Simpson found three certain and three probable cases of syphilis. Syphilitic peritonitis is occasionally found associated with an ulcerative process of the intestinal mucous membrane (*vide infra*). But Preiser believes, that with increased foetal thickening of the peritoneum and effusions in the peritoneal cavity may be easily mistaken for peritonitis. It is possible that in foetal peritonitis, haematogenous septic infections on the part of the mother, play an aetiological part.

The relatively frequent coincidence of foetal peritonitis with atresias and stenosis, and also volvulus of the intestine, points to a causal connection of these conditions. Though it is not yet clear whether peritonitis is the cause of the intestinal obstruction or the latter the cause of peritonitis, Preiser considers that the peritonitis is secondary. Peritonitis with atresias or volvulus is, if present at all, always a local process, confined to the portion of intestine involved. If a volvulus follows an atresia—the occurrence of an intra-uterine volvulus has not been convincingly proved—as a rule, diffuse peritonitis results, spreading over the abdominal cavity and leading to extensive adhesions of the intestine. In a case of diffuse foetal peritonitis M. Unger found two kinks of the intestine (stenoses) above which the meconium had collected, the intestinal walls being very thin. The peritoneal exudate proved sterile. In malformations in the region of the urogenital apparatus, a discharge of urine into the abdominal cavity, and in prenatal spontaneous ruptures of the intestine an extravasation of the contents of the bowels into the peritoneal cavity may result in peritonitis.

Most children with foetal peritonitis die during or immediately after birth; very rarely they survive a few hours or days. The most prominent symptom is the distension of the abdomen, which is visible when the child is being born, caused partly by distended intestinal loops, partly by effusion. The children generally show severe dyspnoea and cyanosis, as the diaphragm is pressed upward and breathing impeded. The symptoms of *ilcus* which occur with longer duration of life come from simultaneously existing obstructions of the lumen of intestine, or from an obstruction resulting from kinks and constrictions of the intestine by peritoneal cords. Peiser considers it possible that localized peritonitis may pursue its course without any sequelae or give rise to symptoms of *ilcus* only in later life.

Only the last mentioned cases are suitable for operative treatment; in severe forms the children die either from the consequences of the simultaneously existing intestinal anomaly or from the disease itself, so that any treatment seems hopeless from the start.

(2) *Acute Peritonitis.*

Acute peritonitis of the newly born is either a manifestation of a general sepsis or it appears as a localized disease. The latter may be of various origins.

Infections arising from the navel, *arteritis umbilicalis*, *omphalitis* or *umbilical gangrene*, &c., may be considered the principal cause of acute peritonitis in the newly born. *Erysipelas* of the abdominal walls originating in the navel or genitals also frequently attacks the peritoneum.

Besides these forms derived from external infection there are also those which arise from the intestine.

Infection of the peritoneum in so-called spontaneous intestinal rupture is unavoidable. In rare cases this occurs pre-natally and may, as already observed, cause foetal peritonitis (Breslau, Clarke, Dohler, Gemersich). A perforation of the small intestine may also occur in some cases, so that Meckel's diverticulum is ruptured or is torn from the navel without its opening being closed. Shukowski connects the rupture with gangrene of the strangulated diverticulum. A. Paltauf attributes intestinal perforation to coprostases, which, owing to exaggerated distension of the intestine causes rupture or necrosis. In perforations existing during intra-uterine life as soon as the infection of the intestinal contents occurs after the birth of the child, acute peritonitis follows. In the majority of cases the spontaneous intestinal rupture probably occurs during birth (Zillner, Falkenheim and Askanaey, Ciechanowski, Sary). It is situated as a rule in the colon, most frequently in the region of the sigmoid flexure, especially when the latter has a long mesentery. According to Zillner, the rupture of the sigmoid loop occurs in this manner: With overfulness of meconium it is compressed between the lumbar portion of the spine or the brim of the

pelvis and on the other side by the abdominal wall; the meconium can pass out into neither the transverse colon nor the rectum, and only slight pressure is sufficient to effect the bursting at the summit of the loop. When the perforation is situated elsewhere congestive processes from kinking of the intestine may be responsible for it.

The life of the child with intestinal rupture is, as a rule, very short; death generally occurs on the second or third day. These children either show no striking symptoms, or they scream very much, refuse nourishment and become extremely cyanotic; as a rule, the abdomen is distended like a drum. In Falkenheim's case, the symptoms of peritonitis appeared only after five days, obviously from partial obstruction of the opening of perforation by calcified meconium; the child lived three weeks.

Infection of the peritoneum from the interior of the intestine probably occurs in the newly born and is of greater significance than is generally supposed. Finkelstein points out that even in the first few days of life peritonitis may arise as the result of ulcers of the stomach and duodenum. Such ulcers are found in particular associated with melana; but this cause of peritonitis must be relatively rare. Apart from infections derived from the navel, possibly the most frequent cause of peritonitis is to be found in enteric infections. In such cases peritonitis may be the result of infectious enteritis, but it also occurs with quite insignificant changes in the intestine, even with an apparently unchanged intestinal mucous membrane.

The author observed a case of streptococcal peritonitis with an hæmorrhagic exudate in a strong child that died on its sixth day; the navel and umbilical vessels at the post-mortem were intact, and the intestine showed no essential changes beyond mild colitis.

If no source of infection is discovered, the likely assumption is that penetration of bacteria through the wall of the intestine has caused the inflammation of the peritoneum.

Circumscribed as well as diffuse acute peritonitis is a frequent sequel of intestinal obstruction such as atresia, stenosis, volvulus, &c. (associated with stagnation of the intestinal contents or affecting the nutrition of the intestinal wall).

Whereas peritonitis used to be considered a particularly frequent disease during the newly born period, it has now become quite rare, like all acute septic diseases of the newly born, thanks to the modern care of the infant and mother during confinement. Earlier observers (Sillermann) describe the clinical picture as follows:

A child in the best of health suddenly develops restlessness, it starts screaming, refuses to take nourishment, and declines. In other cases the disease begins with vomiting, severe diarrhoea, meteorism, tenderness of the abdomen, quick respiration, rapid pulse, and severe prostration. After a few days, in fulminating cases even after a few hours, death occurs with symptoms of collapse,

sometimes with convulsions. But it must be pointed out that vomiting is a frequent, but not a constant symptom, that there is sometimes profuse diarrhea, sometimes persistent constipation, that meteorism may be absent and that an exudate in the abdominal cavity is often very hard to find. The temperature presents nothing characteristic, as in all septic diseases of the newly born. The inconstancy and ambiguity of the symptoms make it appear conceivable that the diagnosis of peritonitis cannot be established with certainty. It should be thought of if a tympanic distension of the abdomen occurs without symptoms of ileus and the child declines rapidly. If in such a case there is any umbilical trouble or erysipelas of the abdominal wall the assumption of peritonitis is probably correct.

The prognosis is hopeless, the occurrence of abortive forms has not been known. Treatment is useless.

(E) Diseases of the Liver and Bile Ducts

(1) DISEASES OF THE HEPATIC PARENCHYMA.

There can be no doubt that in the various diseases of the newly born the liver influences the clinical picture far more than the symptoms and anatomical findings would lead one to suppose. Disorders of the function of the liver, the great chemical laboratory, which is the first to receive the substances assigned for digestion from the intestinal canal, are likely to occur in the newborn child whose intestinal walls exhibit a heightened permeability, and all the more so because the liver of the newly born is perhaps in a state of functional backwardness. Disturbances in the hepatic function may therefore be caused by the relatively small power of resistance of the organ itself, or by toxic and infective substances, which are brought to it from the intestine, or are conveyed by the blood of the umbilical cord. If one considers that every other infection from any source whatever, and every intoxication involve the liver, it is obvious that diseases of the liver during this early period of life must be of the utmost significance, although the number of clinically circumscribed liver diseases of the newly born is very small. The symptoms of so-called hepatic insufficiency are so inconstant, on technical grounds so difficult to investigate, and clinically so indefinite, that it is almost impossible in severe general diseases to recognize the part played by the affected liver in the clinical picture.

As explained in the section on *icterus neonatorum*, there exists in the newly born a certain disposition to icterus in the quality of the liver cells. Skormin enumerates, apart from obstruction in the large bile ducts from congenital diseases, a number of forms of jaundice: icterus septicus, icterus in Winckel's disease, icterus in intestinal infections, and icterus in effusions of blood, icterus catarrhalis, icterus toxicus, icterus with acute atrophy. During the first weeks of life icterus is a very frequent accompanying

symptom of different diseases. Here the common icterus neonatorum is, obviously, chiefly in question, but is influenced, in its intensity and duration, by the general diseases associated with it.

Parenchymatous and fatty degeneration of the liver are quite common accompaniments of all septic diseases. Degenerative processes in the liver are also occasionally found in those diseases in which there is nothing to indicate a bacterial origin, but the effect of a toxin must be assumed that has reached the liver of the fetus by the placenta, e.g., in children of eclamptic mothers that die shortly after birth (Schunorl, Dienst). Dienst points out that the umbilical vein blood containing the eclamptic poison substance passes first through the fetal liver. Hammer examined two bodies of children, post mortem, which, macroscopically, showed no changes, but microscopically exhibited intense degeneration of the parenchymatous organs and the heart. As in the one case the mother had a high fever at the time of confinement, and in the other case there was placental inflammation, it may be assumed that the fatal changes in the infantile organs were caused by toxic substances in the mother's blood.

In earlier literature a few cases have been mentioned of acute atrophy of liver in the newly born. Politzer observed a child that was attacked on its fourth day with jaundice and symptoms of a hæmorrhagic diathesis, and died on its eighteenth day. In the urine post-mortem tyrosin and leucin were found. Aufrecht reports on a child of a healthy mother that died on its sixth day after severe jaundice, extravasations of blood under the skin, and excretion of leucin in the urine. The autopsy showed atrophy of the liver, absence of the regular arrangement of cells, disappearance of the nuclei, in other words coagulation necrosis and extravasations of blood in the parenchyma. There were also bacteria in the liver. Such cases are, of course, to be considered as results of sepsis, but on account of the extraordinary anatomical condition they deserve a special distinction; in many cases in adults bacteria have been found in acute yellow atrophy of the liver, the ætiology of which is still obscure. Bing's case is of special interest. In the child of an eclamptic mother who had had several attacks of convulsions, he found leucin in the urine, and post-mortem severe changes in the liver, consisting of fatty degeneration and hæmorrhagic necrosis.

The changes of the liver in icterus septicus are described by Birck-Hirschfeld as pronounced interstitial and parenchymatous hepatitis. The so-called infective icterus of the newly born, described by Lesage and Demelin, manifested itself clinically in the particular epidemics described by these authorities, in cyanotic attacks, occasionally convulsions and digestive disorders. The symptoms sometimes passed off in a few days, but in other cases led to death. The condition of the liver varied; in the severe cases the liver was red and deliquescent as in acute atrophy, frequently riddled with hæmorrhagic foci. These cases lead to the group of hæmor-

diagnostic diseases, Winckel's disease, &c. A form connected with acute yellow atrophy of the liver is Buhl's disease (*vide infra*). In all these general diseases of infective or toxic origin the symptoms of the liver disease are lost in the picture of general sepsis or intoxication. The liver disease forms here merely an anatomical manifestation in the complex of more severe changes affecting the entire organism.

In spite of its frequent occurrence the pathogenesis of so-called icterus catarrhalis is not yet elucidated. How far a mere duodenal catarrh is present, how far important are infective processes in the liver and bile ducts (cholangitis catarrhalis, &c.) has not yet been established owing to the benign course of the disease and the absence of post-mortem results. By icterus catarrhalis is understood not much more than a clinical symptom-complex, consisting of icterus, acholia of the stools, and bile in the urine, with favourable prognosis. Such forms of icterus are extremely rare in the infant. Randnitz observed a boy of four days with severe icterus, completely colourless, mucous stools and bile in the urine. He died on the ninth day; at the post-mortem the duodenal mucous membrane was found to be swollen and injected. It is unnecessary to discuss whether this case should be classified as catarrhal icterus on account of its malignant course. In any case, in the newly born it is an extremely rare occurrence for pronounced acholic stools to be passed if there is no atresia of the bile ducts. Lovett Morse also mentions a case of icterus catarrhalis in a newly born infant.

Cirrhotic processes in the liver of the newly born child (congenital cirrhosis) are connected on the one side with hereditary syphilis, and on the other side with congenital changes of the bile ducts. In the first case there is a diffuse interstitial hereditary syphilitic hepatitis in the form of hypertrophic cirrhosis (Henoch, Neumann); if there are anomalies of the bile ducts (Freund, Theodor, Thomson, Neukirch) the biliary cirrhosis may be the result of biliary engorgement, or it may be (according to A. Meyer's theory) primary cirrhosis of liver which only attacks the bile ducts secondarily; these are probably inflammatory cirrhoses of toxic origin.

The common forms of cirrhosis of the liver hardly ever occur in the infant, practically never in the newly born.

(2) DISEASES OF THE BILE DUCTS.

Acute inflammation of the bile ducts is extremely rare in the newly born. Wronke describes a case of acute hemorrhagic-diphtheric inflammation of the bile ducts in a child that died on the eighth day.

In isolated cases gallstones have been found in the newly born (Bouisson, Portal, Bärensprung, Still and Thomson); during life severe icterus was observed. Possibly congenital gallstones are the

results of foetal inflammatory processes (Bland-Sutton), products of a congenital disease which finally leads to stenosis and obliteration of the bile ducts.

Atresias and stenoses in the region of the large bile ducts may attack all three ducts, the ductus choledochus, hepaticus and cysticus, or only one of them. A portion of the cases is assuredly of hereditary syphilitic origin. Stenosis of the ducts in these cases comes either from gummatous cholangitis (Kaufmann) or from perihepatitis, affecting Glisson's capsule, and leading to formation of scar tissue, which presses on or constricts the bile ducts (Skorin). This origin appears to be relatively the most rare. Probably it is more frequent for the condition to be similar to the congenital occlusions in the region of the intestine. The bile ducts appear to be partially or entirely obliterated, sometimes transformed into solid cords or impassable just at one spot. Sometimes the ducts, permeable at their commencement, appear to merge into the connective tissue of the ligamentum hepatoduodenale. Finally, there may be total defect of one or two or of all the bile ducts, including the gall-bladder (Theodor). According to Benecke, in the last-mentioned case it is not lack of development or pure aplasia of the bile ducts, but a subsequent obliteration, as embryologically without a bile duct there can be no liver. It is, in fact, an anomaly which Tandler's theory of the origin of intestinal stricture may help to explain (see above). In any case the assumption of an inflammatory origin cannot be altogether rejected, particularly in those cases in which after birth there is not complete absence of bile from the intestine. In these cases therefore total obliteration is not congenital, but only develops gradually during the course of the first weeks of life. It must be assumed here that foetal inflammatory processes, the ætiology of which is still obscure, play some part.

Apart from obstruction by gallstones, an infrequent cause of occlusion of the bile duct is a valvular formation at the distal end of the duct, which may lead to the formation of the extremely rare congenital choledochus cysts (Wettuer).

The most striking symptoms of occlusion of the bile duct is colourlessness of the intestinal contents. As a rule, even the meconium is colourless. In a few cases (Westermann, Ibrahim, &c.) acholia of the stools appeared later, either soon after the evacuation of coloured meconium or during the course of the first few weeks. (In such, an incomplete process of obliteration at birth must be assumed.) The second symptom, resulting from the shutting off the bile from the intestine, is jaundice, which continually increases in intensity and attains such an excessive degree, similar to that observed with malignant neoplasms at the porta hepatis. Jaundice is absent in these rare cases, in which merely the cystic duct and gall-bladder are the seats of disease. Intense bilirubinuria exists corresponding to the jaundice.

All the cases hitherto observed of complete occlusion of the ductus choledochus or hepaticus proved fatal. The children

gradually succumb with extreme cachexia, one reason alone being that the food is not properly digested; the stools are enormously rich in fat. Death is probably due to the consequences of inanition or cholemia, though it is often delayed for many months.

According to the fate of the cases hitherto observed treatment appears to be useless. Operative measures that might be considered are the formation of an anastomosis between the gall bladder or other patent sections of the bile duct (intra-peritoneal bile ducts) and intestine, cholecystenterostomy, or hepato-choleangio-enterostomy (O. Ehrhard). In view of the impossibility of spontaneous recovery an operation *a priori* is by no means to be set aside; it should be undertaken if not during the first few days, in any case as early as possible.

(3) CONGENITAL TUMOURS OF THE LIVER.

Relatively speaking, the most frequent congenital tumour described in literature is sarcoma (round cell sarcoma). In the overwhelming majority of cases reported, there were simultaneous tumours in the suprarenal capsule (de Ruyter, Heaton, Pepper, Bruck, Wilke). The combination of liver and suprarenal tumours independent of one another occurs only congenitally. Probably in this simultaneous development of tumours occurring independently of each other, there is in both organs a suprarenal disease, in which the tumour of the liver comes from an ectopic suprarenal rest. It is obviously a case of hypernephroma.

The disease manifests itself either immediately after birth by distension of the abdomen and a palpable tumour of liver, or the symptoms appear only after three to five weeks, sometimes after a latency of several months. As soon as the tumours have led to perceptible distension of the abdomen, the further growth is generally very rapid and death follows quickly. The development of ascites and jaundice depends on the relation between the tumour and the portal fissure of the liver.

Cases of primary carcinoma of the liver are also known in the newly born (Siebold, Plaut, Yamagiwa). Congenital angiomas of the liver are found in the form of telangiectases, which may involve small areas or a large part of the surface of the liver (Simonini); such a cavernous angioma may burst and may lead to fatal effusion of blood into the abdominal cavity (Hammer), or if multiple nodules are formed, malignant hamangio-endothelioma (Veeder and Austin). Randolp describes a congenital multiple angio-sarcoma of the liver and Ernst a case of "general angiomatosis in the skin and liver of a newly born child."

Hepatic cysts appear in two forms: as simple solitary cysts or as multiple cystoma. The solitary cysts occasionally form large tumours, which may form obstacles to delivery (Witzel, Slinger-Klopp). In other cases they are quite small cysts, probably from

vasa aberrantia, which terminate, not in the hepatic tissue, but in the region of the ligamentum suspensorium and in the suprarenal capsule (Quincke-Hoppe-Seyler); or they are lymph cysts (Shukowski). Such small cysts do not, as a rule, show any symptoms during life.

Echinococcus cysts have also been observed in quite young children (Cruveillier: child of twelve days, Mackenzie). Possibly in these cases infection takes place through the placental circulation.

The multiple cystomata (polycystic disease of the liver, cystic degeneration) are developed from intra-hepatic bile ducts and stand in close connection with adenomata (cyst adenomata). They are probably caused by embryonic disturbances of development in the bile duct system. If the congenital cysts are not large they may remain entirely latent; enlargement may occur in later life.

(4) DISEASES IN THE REGION OF THE HEPATIC VESSELS.

Peripylephlebitis congenita occurs exclusively in syphilitic children and is due to a proliferation of connective tissue round the portal vein and its intra-hepatic branches, or from gummatus infiltration of the bile ducts at the porta of the liver (Chiari, Skormin).

The symptoms of constriction at the porta of the liver are ascites, enlargement of the spleen, gastric and intestinal hæmorrhage; occasionally symptoms of biliary engorgement are also present.

Phlebitis and thrombosis of the portal vein are principally found with phlebitis umbilicalis, in consequence of umbilical sepsis.

Similar reports on a remarkable case of circulatory disturbance in the region of the portal vein, the aetiology of which could not be determined. It was a case of ascites with oedematous infiltration of the lower part of the abdominal wall and development of a collateral circulation in the skin of the abdomen, corresponding to an obstruction of the portal vein, and present from the second day of life; recovery took place after three paracenteses abdominis.

(F) Hernias

CONGENITAL DIAPHRAGMATIC HERNIA.

There are both true diaphragmatic hernias (with hernial sac) and false (without hernial sac); the latter are more frequent in the newly born. The causes of diaphragmatic hernias, according to Liepmann, are the following:

A. Partial or total defect of one side of the diaphragm.

(1) Region of preformed gaps:

(a) *Trigonum lumbocostale*.

(b) *Trigonum sternocostale*.

(2) Other parts of the diaphragm:

(a) *Centrum tendineum*.

(b) *Foramen œsophageum*.

B. Total defect of the diaphragm.

Diaphragmatic hernias are more often left-sided than right-sided. They may remain latent and give the bearer no trouble for years (Winkler). The diagnosis is not easy. According to Grosser, on the discovery of dextro-cardia in the newly born the possibility of diaphragmatic hernia must be taken into consideration. In many cases, especially with severe ruptures, the children die during the first hours of life with symptoms of asphyxia (Lepage, Konow).

HERNIA VENTRALIS LATERALIS.

Lateral abdominal ruptures are in rare instances observed after birth as reducible tumours which are localized either in front of the anterior axillary line or between both axillary lines. They may arise through partial defects of the abdominal muscles (Steinhardt). It is possible there may be disordered development or atrophic conditions owing to pressure on the part by an extremity. In a case of Schenk's the knee of the drawn up leg fitted into the defect. The prognosis of lateral abdominal hernias depends on the size of the rupture. Multiple hernias have also been described. The preliminary treatment in the newly born consists of bandages, followed by a Skoiv truss.

INGUINAL HERNIA.

In about fifty per cent. of all newly born the processus vaginalis is still open at the time of birth and is obliterated only in the course of the first few weeks. In spite of the frequency of this predisposition to hernia, in the actual new-born period few pronounced hernias are found; they only form gradually if the contents of a hernial sac enter the hernial sac through crying and pressure.

We will not enter here into the description and treatment of hernias and will only touch upon the question as to the treatment of inguinal hernia during the first few weeks. Concerning the treatment of inguinal hernia of the infant there are two opposite opinions; on the one hand an early operation is advised, and on the other conservative treatment. In spite of the ideal results of harmless operations achieved through mastery of technique, it must be emphasized that a large number of inguinal hernias in the newly born are cured by simple bandages. Whatever method of treatment may be chosen, an operation must at least not be attempted for the first few weeks. The only treatment to be considered at first for the newly born is that of a truss. Fiedler's woollen truss is recommended as the best for the infant.

UMBILICAL HERNIA.

There is a distinction between congenital and acquired umbilical hernia. The former will be described in umbilical diseases (*vide infra*). By umbilical hernia the acquired form is generally understood. It arises from the umbilical ring not being completely closed and the yielding part of the abdominal wall, situated inside

the ring, being projected under the influence of abdominal muscular pressure and forcing parts of the omentum or the intestine into the hernial sac. The size of the *hernia* is very variable, at first it seldom exceeds that of a hazel nut. The umbilical hernia only occurs when the remainder of the umbilical cord is detached and the wound healed; therefore, strictly speaking, it does not belong to the *neo-born* period.

The treatment consists in the application of a circular plaster bandage after pressing back the contents of the hernial sac between two folds of skin following the direction of the axis of the body. A large majority of infantile umbilical hernias are cured spontaneously by this method, so that an operation is only necessary with very severe hernias.

A very widespread erroneous opinion among mothers, is that by application of a bandage round the umbilical region (the so called binder of a child) umbilical hernia may be prevented. This is not possible as such a bandage has, of course, no influence on the diminution of the umbilical ring and when the disposition is present the hernia will develop in spite of the bandage. After the umbilical wound has healed the application of a bandage is generally superfluous and best omitted, as it may sometimes be irksome to the child.

CHAPTER II.

RESPIRATORY TRACT

(I)—Suspended Animation (Asphyxia)

If immediately after birth respiration is lacking or very deficient, although the child, as demonstrated by the beat of the heart, is alive, the condition is classified as apparent death or asphyxia. "Asphyxia" literally translated means absence of pulse. The expression has been therefore very unsuitably chosen, but it has become part of medical terminology and is generally used as identical with the conception of apparent death. Strictly speaking we are not dealing with a disease, but with a symptom, the *ætiology* of which has no uniformity.

The distinction is generally made between an *intra-uterine* or congenital and *post-partum* or acquired asphyxia. Those conditions are classified as acquired asphyxia which do not result from a disorder of the placental exchange of gases, but only appear after birth, if pulmonary respiration is for some reason obstructed. The causes of this extra-uterine respiratory insufficiency may be central (injury to the respiratory centre) or there may be obstruction in the region of the respiratory passages (malformations, tumours, &c., which compress the trachea, congenital pneumonia, &c.).

In using the term asphyxia, the congenital (acquired during *intra-uterine* life) asphyxia of the newly born is understood. It is

caused by an insufficient supply of oxygen during birth. It may be defined as a process of suffocation not terminating fatally (B. S. Schultz). The obstruction to the supply of oxygen can be traced to two factors: to a filling of the air passages by aspirated substances, or a lowering of the sensitiveness of the respiratory centre from excess of carbonic acid in the blood (Knapp). A sharp distinction between the latter true form of asphyxia and the mechanical asphyxia caused by aspiration is clinically not possible in consideration of the intimate causal connection of both conditions.

Asphyxia of the new-born child nearly always succeeds foetal asphyxia. The placental respiration, the supply of oxygen through the maternal blood, receives a check during the act of birth under physiological conditions owing to labour pains. During the contraction of the uterus the uterine cavity contracts, the placenta, which furthers the exchange of gases, becomes compressed. The obstruction to the exchange of gases is manifested by a slowing of the infant's heart beats, which can be found in almost all cases, particularly in the second stage of labour and during the final labour pains. The compression of the respiratory surface and the retarding of the infant's heart beat do not, as a rule, exceed a certain limit for which Seitz finds the following explanation:—

(1) The established fact of the rarefying of the muscle elements at the site of attachment of the placenta results in the wall of the uterus not contracting at this part with the same energy as the other parts.

(2) Dilatation of the muscular vessels takes place in ordinary muscles during work, and therefore probably in the uterus in labour owing to simultaneous stimulation of their vasodilators.

(3) The rise of blood-pressure in the maternal circulation during labour admits of the penetration of blood into the uterus, in spite of the increase of pressure between the contracting muscular fibres.

The poverty of oxygen and increase of carbonic acid in the blood on the cessation of placental circulation, leads to stimulation of the respiratory centre in the medulla oblongata, which, besides the cutaneous stimulation which appears during the passage from the uterus into the cool air, causes post-partum in a reflex manner the first breath.

Under normal conditions, up till the moment of birth or later, there exists in the blood of the newly born a certain surplus of oxygen. The clinical expression of this condition is the so-called physiological apnoea, which sometimes lasts a few seconds and sometimes several minutes. As a rule the cessation of foetal apnoea occurs simultaneously with the birth of the child, so that an extra-uterine duration of apnoea is not observed. In the large majority of cases respiration starts immediately after the birth of the child. After Caesarean section, in which the intra-uterine circulation is in no way disturbed, occasionally a remarkably long period of apnoea is found, sometimes as long as ten minutes (Knapp). Apnoea and asphyxia must be strictly differentiated one from the other. Apnoea

is absence of breathing from repletion of the blood with oxygen; asphyxia is absence of breathing from obstruction to the exchange of gases and lack of oxygen (Runge).

The causes of absence of respiration, and so of asphyxia resulting from deficiency of oxygen, are of various kinds.

In consequence of abnormally severe and frequent contractions of the uterus, the extreme degree of which is known as "tetanus uteri" (the latter occurs most frequently as a result of inopportune use of ergot preparations), the physiological disturbance of the circulation during labour pains may be converted into a pathological one, so that there is no relief as usually occurs in the intervals between labour pains. If the general intra-uterine pressure which with an unruptured amniotic sac, as under normal conditions, occurs with the same intensity on all sides receives a check, as is particularly likely to occur with more or less complete escape of liquor amni, the interchange of gas may be prevented from a partial increase of pressure in respect of the placenta and umbilical cord. Thus the premature rupture of the fetal membranes may alone lead to asphyxia. It can also happen if the process of labour is retarded after the escape of liquor amni owing to insufficiency of labour pains or pelvic contraction.

The aetiology of the danger of suffocation is most obvious when the conveyance of blood rich in oxygen to the child is disturbed by an obstruction in connection with the umbilical cord. Surrounding of fetal parts with abnormally long cord, partial rupture with abnormally short cord, or complete rupture especially on prolapse of the cord, marginal insertion, pelvic presentation, are relatively frequent causes of death by suffocation during birth or of severe asphyxia after birth.

The placental exchange of gases may, with premature release of the placenta and with placenta prævia, be interrupted or obstructed, and also after arrest of hæmorrhage by bringing down the leg. Here we may also mention the rare cases of birth with a caul, in which the child is born together with the unruptured membranes.

Besides the hindrance to the exchange of gases between mother and child, a distinct diminution of the amount of contained oxygen in the maternal blood may lead to asphyxia in the infant. In particular, cardiac and pulmonary diseases of the mother, diseases of the blood, severe loss of blood, diseases accompanied by severe cachexia, nephritis, eclampsia, and possibly febrile and infectious diseases may also be of significance.

Those asphyxial conditions of the newly born depending on congenital cardiac and pulmonary anomalies do not, strictly speaking, belong to congenital but to "acquired" asphyxia. The same applies to conditions of apparent death in consequence of increase of pressure on the brain and cerebral lesions. Even without cerebral lesions or effusions of blood, trauma of the skull may give rise to apparent death (severe forceps operations and breech extractions through narrow pelvis or rigid soft parts). The increase of intra-

cerebral pressure in these cases has the effect of an insufficient blood supply to the medulla oblongata, and, in particular, to the respiratory centre, and leads to paralysis of the latter. Severe slowing of the pulse may occur from vagus irritation, which is prejudicial to the placental exchange of gases.

Death from suffocation and asphyxia may therefore be caused solely through obstruction to the intra-uterine placental respiration. In the majority of cases a fresh symptom is added, which arises, partly as a consequence of poverty of oxygen in the uterus, partly as a cause of insufficient respiration in the newly-born child, viz., aspiration of liquor amnii from premature respiratory movements.

According to Ahlfeld and the more recent researches of Reifferscheidt, it is probable that the child, even under physiological conditions, performs intra-uterine respiratory movements. Ahlfeld was of opinion that with these respiratory movements the liquor amnii is sucked in as far as the bifurcation and again expelled, whereas Reifferscheidt is of opinion that the respiratory movement take place with the glottis closed, and the power of suction is in any case so slight that the liquor amnii reaches at the utmost the nasal cavity or the entrance to the larynx.

Under normal conditions the respiratory tract is always found to be free of liquor amnii. But if the circulating blood in the fetus is poor in oxygen, the vascosity of the blood may create a stimulus in the respiratory centre and give rise to premature respiratory movements. In births with pelvic presentation and after podalic version, the cutaneous irritation by the external air affecting the lower part of the body may stimulate the respiratory centre. The consequence is that liquor amnii, mucus, vaginal secretion, blood, or meconium, owing to increased peristalsis, may be drawn into the trachea. If the child has left the genital passages the aspirated substances may, partially or totally, obstruct the entrance of air and create a new obstruction to the increased necessity of oxygen. Even before the exit of the child fresh dangers develop through aspiration; with the inspiratory movement of the thorax the pulmonary circulation develops, the lungs aspire blood from the right heart, and withdraw it from the already insufficient placental circulation. Hence, aspiratory movement may be both cause and effect of asphyxial conditions.

DIAGNOSES AND CLINICAL SYMPTOMS OF ASPHYXIA.

The obstetrician recognizes the menacing asphyxia chiefly by the gradual slowing of the infantile cardiac sounds, and in particular by the prolongation of bradycardia during the pauses of labour. When paralysis of the vagus centre sets in the action of the heart may also become irregular. A further important sign of intra-uterine asphyxia is the admixture of meconium in the escaping liquor amnii. It points to increased intestinal peristalsis, such as

happens with every asphyxia from hypervæmosity of the blood. Premature respiratory movements may be detected by twitchings of the visible and palpable skull (Altfeld), also on auscultation by peculiar sounds derived from twitchings of the body (Seitz). Vagusæmia, which occurs in rare cases, represents nothing more or less than intra-uterine respiratory movement. Finally, a particularly loud umbilical souffle, increasing in distinctness, indicates that the child's life is in danger; excessive foetal movements may also be regarded in the same sense.

One generally differentiates between two degrees of apparent death: asphyxia of the first degree, blue asphyxia (*asphyxia livida*), and asphyxia of the second degree, pale asphyxia (*asphyxia pallida*).

With asphyxia of the first degree the skin is dark red or cyanotic, the extremities appear to be somewhat protruded, and the conjunctivæ injected. The reflex excitability of the skin is maintained, the heart beats slowly but strongly, the tonicity of the muscular apparatus of the body is present, in fact it may be increased. The child does not breathe or only insufficiently, with perhaps only a few superficial, gasping inspirations. At this stage, after some simple efforts of resuscitation or even spontaneously, respiration starts within a few minutes. The prognosis of blue asphyxia is accordingly favourable.

The so-called pale asphyxia constitutes the more severe degree of asphyxia. Owing to irritation of the vasomotor centres from the blood being overlaid with carbonic acid gas, there is in this case contraction of the cutaneous vessels; the blood is driven into the interior of the body and presents another opposition to the weakened heart. In consequence the skin appears extremely pale, the visible mucous membranes are livid. The reflex response to stimulus is absent, as is easily found by putting the little finger into the child's throat. The action of the heart is either slowed, or owing to paralysis of the vagus centre increased; the cardiac sounds are dull and soft. According to Schultze, the characteristic difference between both forms of apparent death consists in the contrast of the tonicity of the muscles. With pale asphyxia it is either lowered or completely lacking, all the limbs hang loosely from the body. The condition of the child resembles that of a dying or dead person; only the weak action of the heart and isolated respiratory movements point to the fact that death is still in abeyance.

Though both extreme forms of apparent death are easily distinguishable, there are also transitional forms. From the pathological standpoint Altfeld considers that a distinction between the two forms is out of date and quite superfluous. Nevertheless the titles, *asphyxia pallida* and *livida*, as clinical types are justified inasmuch as they present certain guides to treatment.

The prognosis of grave asphyxia is very serious. If the child is left to itself, life is gradually and completely extinguished. If, after efforts of resuscitation, the paleness of the body disappears, and the action of the heart becomes stronger, the outlook is favourable. But if after half to one hour's effort to restore life the

child does not breathe freely, the prospect of a favourable issue may be considered hopeless. And even if the heart beats for some time, death generally supervenes. Nevertheless, as children have been known to recover after many hours of effort to restore life, the latter must be continued as long as a heart beat is audible; hope must not disappear till the heart has stopped.

TREATMENT.

The chief object is to convey oxygen to the impoverished tissue, to stimulate the respiratory centre, and to improve the failing circulation.

The methods which aim at conveying oxygen by extra-pulmonary means, have had no satisfactory results. Infusions of oxygen into the umbilical veins have been undertaken in the following manner: A metal syringe, provided with a cock, is filled with oxygen, the advanced piston piercing the india-rubber tube attached to an oxygen cylinder, so that the piston is forced back through the gas streaming in. The cock is closed and the syringe, provided with a blunt sterilized nozzle, is tied into the umbilical vein after the umbilical ligature has been loosened, so to 12 c.cm. of oxygen are then injected in the course of a few minutes (Sultz, Offergold). The method is not considered to be very promising, and is not altogether without danger (cardiac dilatation).

The more natural way of supplying oxygen is to convey oxygen or oxygenized air to the body through the lungs. This is accomplished by trying to induce respiration or by performing artificial respiratory movements, or blowing air or oxygen straight into the respiratory passages.

The primary condition for obtaining a favourable result is to free the passage. All aspired substances must be—as far as possible—removed from the air passages. The mucus collected in the mouth and throat can be removed by the little finger (wrapped in a piece of sterilized muslin or gauze). To do this it is best to hold the child head downwards by the legs, in order to facilitate the discharge of secretion. The nasal cavity can be cleansed by a soft feather or a small wad. Aspiration by means of a catheter is hardly necessary to remove substances in the nasopharynx. In order to remove substances from the trachea and bronchi, a tracheal catheter (a common elastic catheter about 3 mm. in diameter) must be introduced through the larynx, a procedure simple enough for a person skilled in intubation, but one that must be learnt. The suction of the obstructing material is done with the mouth, or better still with a small rubber ball. It is advisable to insert a small glass recipient which, as suggested by Knapp, is provided with an outlet which can be closed by a pinch cock. With Schultze's method the use of the tracheal catheter is considered superfluous.

Reflex Stimulation of Respiration by Irritation of the Skin or Mucous Membranes.

In mild forms of asphyxia the stimulation of the skin by the air is often sufficient to excite the respiratory centre. If the child does not begin to breathe after one or two minutes the smacking of the buttocks or sprinkling of cold water over the body will generally produce the first inspiration. If more energetic methods must be resorted to, the body should be rubbed with warm towels or with the hand, or the child should be dipped into cold water. An excellent method of resuscitating asphyxiated children is the warm or hot bath (38° to 40° C.; 100.4° to 104° F.). Ahlfield prefers this procedure to any other, even to methods of artificial respiration. In a hot bath the skin capillaries are dilated, the spasm of the vaso-constrictors is relaxed, and the internal organs overfilled with blood, particularly the heart, are relieved. From this point of view an oxygen or mustard bath might be recommended, also mustard bandages. Ahlfield considers that all asphyxiated children, that have the capacity to live, need merely to have the air passages freed and a warm bath, in order to be able to breathe freely, cutaneous irritants shortening the period of deficient respiration. Indirect stimulation of the respiratory centre by cutaneous irritation is decidedly superior to direct stimulation (subcutaneous injections of ether, camphor, caffeine) though the latter may be used as a subsidiary measure, especially as it may be an effectual means of reviving the action of the heart. Further cutaneous stimuli with good results, apart from vigorous massage, are cold douches or a quick dip into cold water, during the warm bath.

Nacke recommends heart massage by rubbing the cardiac region with a springing stroke about 300 times a minute. The heart very often recovers even if the cardiac sounds are hardly audible with the stethoscope.

Methods of Artificial Respiration.

As Seitz remarks, the number of methods of artificial respiration is legion, as many authorities appear to make it their ambition to discover new methods of resuscitation. These are very often merely trifling modifications. Detailed descriptions of the various methods have been given by Knapp, Seitz and Runge. Only the most important will be described here.

Marshall Hall's method. The asphyxiated child is laid face downwards and one or both arms placed under the forehead, the chest being propped up by means of a towel. After a few seconds the body is slowly turned on one side, then moved quickly back again to the first position. The rotations must be continuously and evenly repeated about fifteen times a minute. Whilst the child is on its stomach slight pressure and massage are to be made along the back.

Sylvester's method is the same as that used for the resuscitation of adults: the arms are drawn up over the head, then downwards and the thorax firmly pressed. Instead of the arms the shoulders may be drawn from in front or behind. The child is laid on its back, the shoulders placed somewhat higher. The tongue must be drawn forward in order to give air free access. The child must be held firmly, which is best done by an assistant holding the legs.

Prochownik's method may be used before detachment of the umbilical cord. With one hand the child is held by the legs in a vertical position. With the other hand, or if there is some one else to hold the child, with both hands the thorax is so clasped, that the thumbs and balls of the thumbs rest on the sternum and the fingers on the back; rhythmical compressions are then made, in which the first compression for removing the aspired substances should be specially vigorous.

Schultze's method is that most commonly used by obstetricians. Runge describes it in the following manner:

"The child is held with both hands, by the shoulders, the thumbs being placed on the front surface of the thorax, the fore-fingers in the armpits and the other fingers obliquely on the dorsal aspect of the thorax. In this way the head is supported by the ulnar margins of the wrist joints. The operator then places himself with outstretched legs, holds the child in the aforesaid manner and casts a glance at the ceiling in order to estimate its height. From this hanging position the child is then slowly swung upwards, so that by slight elevation above the horizontal position the lower part of the body gradually sinks down upon the upper part, the thorax is not allowed to be compressed by the fingers in any way. This results in a considerable pressure on the organs of the thorax as well as the diaphragm and the other chest walls, in fact a passive expiratory movement. In consequence, the aspired fluids are forced—especially if the child is slightly shaken—out of the mouth and nose, not infrequently into the face of the operator. After a short pause of three or four seconds the body of the child is swung downwards. The thorax, now free from any pressure, expands on account of its elasticity, the ribs are raised, and the diaphragm moves downwards. This results in a purely passive, extensive inspiration, and as a rule the air is driven audibly through the glottis into the air passages. After a pause of a few seconds the child is again swung upwards and downwards, this procedure being repeated six to eight times. The child is then placed for a few minutes in a warm bath, in order to avoid cooling down too suddenly, and the effect of the passive respiration can then be observed. If the respiratory movements have not started, the swinging must be continued."

The method can only fulfil its purpose if carried out exactly, and any carelessness may be most detrimental to the child. It must therefore be practised. Before the swinging is begun the root of the tongue must be firmly pressed forward, so that the epiglottis is situated upwards.

Gaszynski's method: The child is placed on the doctor's knee, its head hanging slightly downwards. The right hand seizes both feet, and the left hand both the hands. (Forefingers between both hands, the thumb seizing the right hand, the other fingers the left hand; feet somewhat raised, head slightly downwards.)

Expiration: The left hand, together with the child's hands, is approximated to the front surface of the thorax, and pressure is exercised on the upper front surface of the thorax in a dorsal direction and to the side of the abdomen. Simultaneously the feet are drawn towards the thorax, the body being bent in the lumbar region. **Inspiration:** Hands and feet are placed straight.

Inspiratory and expiratory movements are repeated about twenty times a minute. If the tongue sinks backwards it must be held with a Pean's forceps.

Dew's method: Neck between thumb and forefinger of the left hand. Head downwards. The upper part of the back lies in the hand. With the right hand the knees are held between the thumb and fingers, so that the thigh lies on the palm of the hand. Inspiration is produced by pressing down the pelvis and lower extremities. With expiration the head is pressed forward to the thorax, the thighs drawn up to the abdomen. The expiratory movement should always be undertaken first.

Ogawa's method: (1) *Method of Slapping.*—The back of the child is placed on the outstretched hand, with the head and extremities hanging downwards. The trunk is thereby stretched, and the chest is arched. With the closed finger-tips of the other hand the cardiac region should be hit lightly and evenly about ten to fifteen times a minute, and the finger-tips should spring back immediately after each contact with the body. This method is not only a cutaneous irritant, but also produces expiration. After each contact with the finger-tips the thorax springs back to its original position.

(2) *Method of Swinging.*—The child lying on its back is seized by the feet, so that the hand clasps the ankles from behind, the forefinger lying between both internal malleoli, the thumb on the external malleolus of one foot and the other fingers on the external malleolus of the other. The other hand is pushed under the head towards the child's back, the thumb and forefinger act as a lever under the neck, the other fingers span the chest. The palm of the hand supports the back. The hand must hold the child firmly without exercising any pressure on the chest. The child thus held must be lifted to the height of the chest. The hand holding the head end pushes the upper part of the body slowly upwards, then downwards towards the feet till the face touches the back of the feet. At the same time the hand at the back exercises pressure on the upper part of the body. This bending of the child can be carried on till the head hangs down, and the gluteal region is arched. With this movement expiration is produced. The blood column in the aorta is set in motion, and fluid is forced out of the air passages. After a few seconds the inspiratory

position should be once more resorted to, and in some cases above the horizontal position. Then again, after a few seconds, the expiratory position, and so forth. If a still more vigorous procedure is necessary, the child, in the inspiratory position, must lose its support at the head by the removal of the hands and only be held firmly at the feet, so that the arms dangle over the head. The swinging must be repeated with regular pauses eight to ten times a minute.

Sokolow's method: The child is laid on its back on a table, the head hanging over the edge. The doctor, standing on the right side of the child, supports with his left hand the head and bends it backwards and forwards. With the right hand he takes hold of the legs and presses the knees towards the abdomen, and then stretches them out again. The first movement, that of pressing the knees towards the abdomen, must coincide with the movement of the sternal flexion of the head.

Laborde's method of rhythmical tongue traction has not received much recognition. It consists of gently pulling the tongue between the fingers about twenty to thirty times a minute. By this means the glosso-pharyngeal nerve is supposed to be stimulated.

An attempt has been made to induce respiration by electric stimulation of the phrenic nerve, but this method has not been generally adopted.

Insufflations of Air and Oxygen.

The most primitive method is the direct blowing from mouth to mouth. It is best undertaken with the nose open instead of closed. After each insufflation the thorax should be compressed, by gentle manipulation. The effectiveness of this treatment is diminished by a considerable quantity of the insufflated air getting into the œsophagus instead of the trachea. On account of the danger of infection the method is not altogether to be recommended.

Insufflation by means of a catheter appears to be more suitable. According to Olshausen, who warmly recommends the method, the object of insufflation is less the supply of oxygen as the removal of atelectasis and the improvement of the heart's action, which is often manifested by the acceleration and increasing strength of the cardiac contractions, even after six insufflations. As air sometimes escapes from the catheter, Ribemond has provided the so-called *Chaussier-Depault* tube with an olivary thickening and with a corresponding curve. The ball made of indiarubber from which the air is insufflated contains about 28 c.cm. of air, as 30 c.cm. represents the largest amount of admissible quantity of air.

It has also been recommended before insufflation to perform intubation or tracheostomy; but in consideration of the very unfavourable prognosis of the operation in the newly born tracheotomy should not be employed.

Instead of atmospheric air pure oxygen may be insufflated.

Knapp conducts the oxygen by means of a rubber tube, either to the mouth or nose of the child, one or the other being held. Zangemeister insufflates it with a tracheal catheter, by means of pressure on a thin rubber ball, straight into the trachea, which has been cleared of mucus. After the lung has been slowly distended careful pressure is exercised on the thorax, whereby the air escapes together with the tracheal catheter, which must not be too thick. Owing to the thinness of the rubber ball it is easy to avoid over-pressure, thus minimizing the danger of emphysema.

Bucura combines the supply of oxygen or insufflations of air to the child's mouth with artificial respiration by Silvester's method, whereby the oxygen reaches the lung through the mouth.

Hoerder recommends a modification of the so-called "over-pressure apparatus" of Brat and Schneider. The method makes it possible with the right pressure to keep the lung expanded during the phase of expiration, without obstructing the retraction required for ventilation of the lung. The over-pressure is produced by impeding the breathing by the application of an expiration valve. This method is carried out by a tracheal catheter, marked so that it can be introduced to a little above the bifurcation. First the mucus is aspirated into a small glass bowl and then oxygen insufflated. The pressure is regulated by a water valve. The pumping of oxygen (about thirty to forty times a minute) is only to be stopped when the child has obtained a healthy colour, and the catheter only removed when the child breathes regularly and spontaneously. Artificial respiration by means of this apparatus is supposed to act better if the child's breathing has completely stopped.

Engelmann's method of insufflation of oxygen at a pressure aims at expansion of the infantile lung without catheterization of the trachea. Von Tiegel's apparatus consists of an ordinary oxygen cylinder with a simple water valve and a suitable face mask. This is so constructed that it can be applied to the face by means of a hermetically closing rubber ring. A relatively large rubber ball is screwed to the top of the mask, and communicates with it through a wide opening. A piece of tube is attached to the interior of the mask, and through this the stream of gas is conducted from the cylinder to the ball. From here it is partly inspired, and is partly mixed with the expired air, together with which it passes through an outlet in the lower part of the mask into a tube, and from thence into a water pressure regulator. This consists of a simple metal tube that is placed in a receptacle filled with water and provided with a centimetre scale. According to the depth of immersion of the metal tube the pressure can be varied in the whole system to a desired point. Quite a small pressure (2 to 3 cm. water) is sufficient to start respiration.

The question: which of the numerous methods is to be chosen in individual cases? is very hard to answer. The views held by

obstetricians are conflicting. It is essential to know exactly the object of the method chosen, and to be ready for any emergency necessitating quick action on the part of the medical attendant.

In mild forms of asphyxia quite simple methods usually suffice (cleansing of air passages, cutaneous stimulation, warm bath). If the object is not attained, or if it is a case of more severe asphyxia, a more vigorous method must be chosen. Schultze himself recommends his own method only in the second degree of apparent death, that which is combined with flaccidity of the muscles.

Which procedure to adopt, whether the swinging or another method of artificial respiration, whether air or oxygen insufflations, depends on existing local conditions, and particularly on complete command of the technique of the method to be used. It is universally acknowledged that some methods, particularly Schultze's, if awkwardly carried out by an unskilled person do more harm than good.

Whether an asphyxiated child should have the umbilical cord ligatured immediately or later is another disputed point. In the desire to delay the ligature and division of the cord it is calculated that, with the umbilical cord still pulsating, the possibility exists of placental blood, rich in oxygen, being conveyed to the child, whereas, on the other hand, later ligature is considered to be useless. Seitz considers that the period which elapses before one can estimate the degree of asphyxia is quite sufficient. As long as the child is lying between the mother's legs, apart from cutaneous stimulation, the only method possible is that of Prochownik. Whether the clearing of the air passages should be undertaken before ligature and division or in the bath seems quite immaterial.

During ligature of the cord some have recommended that about a spoonful of blood should be allowed to escape from its cut surface in order to unburden the overfilled heart. In any case the measure is harmless, if superfluous.

If radical methods for resuscitation can be avoided they must be withheld. It is not expedient to swing every stillborn child or to insufflate air; for both methods, even when carried out correctly, are not altogether without danger.

Discussions on the dangers of Schultze's method take a considerable place in obstetrical literature. Premature birth, intracranial hæmorrhage, and fractures of large bones are contra-indications. It must be remarked that the diagnosis of intracranial hæmorrhage, also of fractures, in particular of clavicular fractures, is not so easy to establish, and that there is no time for long differential diagnostic considerations. With a collar-bone fracture, if, as is usual, there is no severe dislocation of the fractured ends, the possibility of injury to the pleura or lungs is hardly to be feared if the swinging has been correctly performed. Kehler deprecates the swinging *a priori* after severe forceps operation.

Schultze's method has been made particularly responsible for the existence of internal hæmorrhages (liver, suprarenal capsule,

spleen, lung, vertebral canal, &c.); for hæmatoma of the sternomastoid and oblique paralyzes (Abfeld, Hengge, Bureklund, Ogata, and others). Schultze and his followers have replied, quite justifiably, that hæmorrhages have often been observed without previous swinging, and that they should be considered more as the results of asphyxia than of efforts of resuscitation. It is, of course, possible that with the great frequency of small extravasations of blood, in consequence of asphyxial conditions, the swinging increases the size of the ruptures, and that slight hæmorrhages may thereby become more extensive. If internal injuries at birth are present, creating severe extravasations of blood, as, e.g., in intracranial and suprarenal capsular hæmorrhages, every procedure to improve the circulation harbours the danger of severe secondary hæmorrhage, in particular such a procedure as Schultze's method. M. Hirsch believes that a considerable number of deaths during the first week is due to the consequences of efforts at resuscitation. This, it need hardly be said, is difficult to prove. However, on the basis of all these considerations the logical conclusion is, that this swinging method should only be performed if it cannot possibly be avoided. If it is considered indispensable the possibility of unpleasant consequences must be risked.

The danger of insufflation of air lies in the possibility of rupture of the lung and formation of emphysema. No brutal trauma is necessary, for the elastic tissue in the lung of the newly born is very weak in resistance, and not even fully developed in premature children (Teuffel). Therefore it is wiser to choose those methods, permitting the introduction of an exactly measured quantity of gas rather than mere insufflation, in which the strength of pressure is not easy to gauge.

CONSEQUENCES OF ASPHYXIA.

The post-mortem lesions in children dying in an asphyxiated condition correspond with those found in death from suffocation. They consist essentially in the results of stasis; the heart, particularly the right half, and the large vessels of the thorax are distended with blood; the brain and organs of the thoracic and abdominal cavities are plethoric and oedematous. In nearly all the organs, as well as in the skin and mucous membranes, more or less numerous extravasations of blood are found (ecchymoses from suffocation). In milder forms of asphyxia petechiæ are found, particularly in the skin of the head, also in the conjunctivæ (they are also to be found after every protracted labour, without a marked asphyxial condition resulting), in severe forms chiefly in the internal organs and especially in the pleura. The peritoneal, pleural and pericardial cavities may contain serous extravasations. If aspiration has taken place the aspirated substances will be found in the trachea, bronchi and their branches.

These pathological conditions which are met with in the dead body indicate what changes may also be supposed to occur in the

surviving child. The results of asphyxia therefore are those of stasis, hæmorrhage due to passive congestion, and aspiration.

Even if the resuscitation of the child is successful, 20 to 30 per cent. of stillborn children succumb to sequelæ (Senz). The mortality is highest in the first two days. According to Poppel, within the first week nearly seven times as many stillborn resuscitated children die as those born normally. The mortality stands, therefore, in exact proportion to the duration and severity of the asphyxial condition.

The causes of death are not uniform, and it is not always easy to separate the real consequence of lack of oxygen and carbonic acid intoxication from accidental diseases. Asphyxial attacks may be repeated, even if the child has breathed and cried after the first efforts at resuscitation. This may be due to insufficient stimulation of the respiratory centre. In the majority of cases it is due to atelectasis from insufficient expansion of the lungs or obstruction of the bronchus by aspirated substances. In such cases the efforts must be repeated and artificial respiration, baths, and douches renewed. As has been observed in premature cases such children may succumb in spite of everything, owing to insufficient stimulation of the respiratory centre, which manifests itself in deficient respiration and in attacks of cyanosis. Added to these is the danger of inflammatory lung complications in atelectatic parts of the lung, and after aspiration of liquor amnii, especially if the latter contains germs.

In the first few days of life children born asphyxiated often show remarkable apathy; they hardly cry at all and take nourishment badly. In spite of favourable conditions it is often impossible to make these children suckle, and they must sometimes be fed from the bottle or even with a spoon. These symptoms may pass off in the course of a few days. They are probably symptoms resulting from venous congestion of the cranial contents, from hyperæmia and œdema of the brain and leptomeninges.

It may be considered a fact that children born asphyxiated frequently exhibit cerebral symptoms sooner or later. In many cases these are not the pure consequences of asphyxia, i.e., of poverty of oxygen and excess of carbonic acid gas, but of cerebral lesions (intracranial hæmatoma, contuso cerebri) occurring at the time of birth. In such cases asphyxia is co-ordinate with cerebral lesions or the result of such, but not the cause; it must therefore, anamnestically, not be considered as such, but as the indicator of a severe process of labour. It is often extremely difficult to decide which is the real result of asphyxia and which the result of birth trauma.

Post-partum asphyxia is attributed by many neurologists and psychiatrists as of great importance in the occurrence of certain arrests of development, partly in the motor and partly in the psychic sphere. Association with idiocy has also been conjectured. Schultze is of the opinion that permanent injury is only to be feared in protracted asphyxial conditions lasting many days. If

after two hours the child is made to breathe there is no danger to its mental development. In this respect Hanne's statistical researches are interesting.

Among 150 children born in an asphyxial state, 3.2 per cent. were mentally abnormal, 0 per cent. idiots; among 150 children born by operative measures but not asphyxiated, 3.3 per cent. were mentally abnormal, 1.1 per cent. idiots; among 150 children born spontaneously and entirely normally, 3.4 per cent. were mentally abnormal, 1.1 per cent. idiots. Hanne concludes from her researches that the question as to whether a difficult and asphyxial birth tends more to abnormal development than a normal birth must be answered in the negative, but whether with justification further statistics must prove.

(II) Diseases of the Upper Air Passages.

(A) Diseases of the Nose and Nasopharynx.

Among the malformations which may involve the external nostrils, the nasal passages and the region of the choanae, those that affect the last are practically the most important. Congenital closure of the posterior nares may be unilateral or bilateral, membranous or bony (Boulay). If it is bilateral obstruction to respiration may occur, thus giving rise to asphyxial attacks. If one closes the mouth inspiratory contractions of the cheeks are noticed. The children suck badly, keep the mouth open, they snore, and sometimes have asphyxial attacks. The closing of one choana may long pass unnoticed. With severe obstruction to respiration an attempt may be made to break through the obstruction with a probe or trocar and then dilate it gradually.

The nasal cavity of the newly born is characterized by considerable narrowing of the nasal passages. The lower turbinate is situated close to the lateral wall, so that the lower nasal meatus under physiological conditions is very narrow. The continuation of the nasal passages into the pharynx has the form of a rectangular tube (Tondoy).

The narrowness of the nasal passages gives rise in occasionally quite healthy children to a snuffling noise heard during inspiration. Congestive swellings of the nasal mucous membrane may increase the stenosis. The knowledge of this harmless aetiology of snuffles is important, in order to avoid diagnostic errors, principally confusion with syphilitic rhinitis.

Apart from the most important form of rhinitis, viz., syphilitic (*vide infra*), various other inflammatory diseases occur in the newly born. Infection of the nasal mucous membrane may occur at birth during the passage through the maternal vagina. But in general these kinds of vaginal infections are not frequent. The bath water may also be a source of infection. It is questionable whether any importance can be attached to loss of heat and inhalation of cold air as a disposing factor. Various pus-cocci, also gonococci and

diphtheria bacilli, may be the causes of nasal discharge (*vide infra*). Congenital rhinitis is nearly always of syphilitic origin. The symptoms of coryza appear generally one or two days after birth. The anatomical conditions are such that relatively insignificant swelling and collections of secretion may lead to closure of the nasal passages. The child has difficulty both in breathing and sucking. Torday points out that in such cases, when sleeping with the mouth open the tongue lies up against the palate, and therefore the oral cavity may also close, tending to produce attacks of dyspnoea and cyanosis. Dyspnoea may also be caused by an extension backwards of the swelling of the mucous membrane, and consequently disturbance in the co-ordination of the musculature of the upper part of the pharynx and soft palate. Further danger threatens the child through spreading of the inflammation to the lower air passages. A septic state may also arise from disease of the nose. Baginsky observed several cases of sudden death among newly born infants with severe coryza.

As in the period of infancy in general, the rule, that the child must be protected as far as possible from infectious surroundings, applies particularly to newborn children of small powers of resistance against aerogenous infections. A perfectly harmless catarrhal affection of the air passages in an adult, may be extremely detrimental to the child. If the mother, nursing the child, has any such affection, she should take care not to breathe on the child. She should wear a face mask or protect the child from droplet infection by means of a cloth laid on its face. It is more difficult to avoid vaginal infection. If the mother is suffering from leucorrhoea, the nose of the child should be cleansed immediately after birth with a small wad of cotton wool, and particularly if aspiration has already taken place.

If rhinitis is already present the most efficacious treatment is that of ointment (3 per cent. boracic lanoline) applied far up the nostrils. Instillation of a 1 per cent. silver nitrate solution, or 3 per cent. H_2O_2 is also of value; the latter is not only effectual as a disinfectant but acts as a mechanical purifier by formation of foam. Stern recommends the use of an elastic catheter in the nose to drain off the secretion. The application of adrenalin is strongly recommended for inflammatory conditions (cotton wool soaked in 1 per 1,000 solution for a few minutes every three to four hours).

The feeding of a newborn child with nasal catarrh often creates difficulties. If feeding is sometimes difficult for older infants, it is doubly so for a child unpractised in the technique of sucking and which frequently has to take from a poorly secreting breast. The snuffles may form a very undesirable obstruction to feeding necessitating from time to time feeding with milk which has been drawn off.

DISEASES OF THE ACCESSORY SINUSES.

Suppuration within the maxillary antrum is an exceedingly rare condition in infancy. Cantestro has reported eight cases in the

newborns. The infection within the antrum usually originates in the nasal route—exceptionally in the skin or in the jaw—and the symptoms consist of reddening and swelling of the cheek and lower lid, of the gum and palatal arch, protrusion of the ball of the eye; in course of time an abscess may form and sinuses develop. Muggia has seen a case of suppurative inflammation of the maxillary antrum in an infant twelve days old; the patient eventually died of pyæmia. In his opinion, this form of inflammatory process often remains latent for a long time, and escapes recognition because of other coincident disease in the patient. In Finkelstein's view, the cases diagnosed as "Empyema of the Antrum of Highmore" were really instances of disease of the bone or osteomyelitis.

DISEASES OF THE PHARYNGEAL TONSIL.

The process of catarrhal inflammation of the anterior nares is apt to extend to the pharyngeal tonsil, and to the adenoid tissue of the roof of the pharynx. The latter is of greater importance as the seat of morbid processes of infancy than the adenoid tissue of the palatal tonsils, *v. p. 229*). Posterior rhinitis or retro-nasal angina is a frequent complication of the more severe forms of coryza, and to a large extent is responsible for disturbance in breathing and the taking of nourishment. The disease is manifested by the mouth being constantly open, and the snoring and rattling sounds on breathing. Erdely has occasionally succeeded in demonstrating the enlargement of the pharyngeal tonsil in young infants by palpation of the soft palate and feeling the uneven surface of the tonsil through it. The lymphatic glands at the angle of jaw are liable to swell in this condition.

Owing to the concealed position of this tonsil direct treatment is not easy. The most rational treatment is the use of a spray of H_2O , directed towards the pharyngeal space, and, if possible, against the roof of the pharynx.

Although temporary enlargements of the pharyngeal tonsil, as a result of inflammatory processes, have been observed in very young infants, there has been no proof of the existence of congenital hypertrophy of non-inflammatory nature, *i.e.*, adenoid vegetation, in newborn infants. The pathological investigations of Czerny and Bartenstein on this matter yielded a negative result. Czerny holds that the origin of true adenoid vegetation depends upon the exudative diathesis, and that they may be checked by an appropriate nutritive regimen and other treatment. Hyperplasia of the pharyngeal tonsil is, however, not an early symptom of the exudative diathesis.

If the catarrhal inflammation of the nose or the naso-pharyngeal wall spreads downwards, causing pharyngitis, laryngitis, or tracheitis, hoarseness and cough may supervene. Symptoms of respiratory obstruction may occur occasionally. In regard to otitis, *vide infra*,

(B) Diseases of the Upper Air Passages accompanied by Stridor.

The symptoms of stridor may be produced by many causes in early infancy, just as at a later age. The term congenital stridor does not connote any definite disease; it merely signifies a symptom. Stridor occurs when there is obstruction in the region of the respiratory tract; it may be in the laryngo-tracheal tube, or it may be outside the trachea leading to its compression. But when the term "congenital stridor" is employed it is customary to exclude the known factors of symptomatic stridor, e.g., the sequela of a congenital goitre. There is, however, one variety of stridor dependent upon a demonstrable respiratory obstruction for which the term "congenital stridor" is still in vogue, viz., the tracheal stenosis due to pressure of the thymus gland. The part played by the thymus in the causation of congenital stridor is still a subject of controversy. Some authorities always attribute congenital stridor to compression by the thymus, and therefore call the condition "thymic stridor," while others deny the general responsibility of this gland in the etiology of this condition, and they refer the cause of the ordinary congenital stridor to the larynx. The two views are, however, not mutually exclusive; it may be assumed justifiably that the term "congenital stridor" embraces two forms, a laryngeal stridor and a thymic stridor (*stridor inspiratorius neonatorum*, *laryngismus stridulus neonatorum*).

(A) CONGENITAL INSPIRATORY LARYNGEAL STRIDOR (BENIGNS).

Inspiration is sometimes accompanied in infants by a peculiar high-pitched noise which resembles the crowing of a cock or an excited hen (Feet), or a clucking sound, or hiccough (Finkelslein). This may occur on the very first day or during the first week; but not, as a rule, later. It is not the same kind of noise which occurs in the crowing of laryngeal spasms, or in the whoop of pertussis. It is very different to the hoarse sound heard in the obstruction of laryngeal diphtheria and to the stridor which is a feature of congenital goitre. Heubner compares it to "the sibilant rhoncha heard in the expiratory phase of respiration in asthma, but yet is not quite like it." Sometimes this regular prolonged sound is audible with each inspiration—even in sleep, when it is occasionally more intense; in other cases the loudness of the sound varies, and it may only occur when the infant is excited, and subsides during sleep. As a rule, the expiration is perfectly quiet. Slight inspiratory retraction is frequently seen in the neck, epigastrium, and in the lateral walls of the thorax. The infant does not appear to suffer in any way from this difficulty in breathing. The aspect and general condition are usually quite good, and cyanotic discoloration or dyspnoea are very exceptional eventualities.

This condition usually persists for a few weeks or months, and then gradually disappears without leaving any kind of sequela. All

trace of stridor has vanished by the end of the first year. The prognosis is therefore, as a rule, quite favourable, and is only unfavourable if any complication occurs in the respiratory organs. If a child, suffering from this affection, is attacked by rhinitis, laryngitis, or bronchitis, a state of dyspnoea is very liable to supervene, and this may become considerably aggravated as the illness progresses. Sudden death has been recorded in cases of infants with congenital stridor, but it may be doubted whether these were really instances of the benign form. It is true that the clinical picture of congenital stridor is quite characteristic, but it must not be forgotten that more serious diseases may occasionally masquerade under the guise of a harmless stridor. (*vide infra*.)

Most paediatrists are unanimous in referring the stridor now under discussion to a laryngeal origin. It is therefore desirable to speak of it as *laryngeal stridor* (or rather, as *inspiratory laryngeal stridor* because the adventitious sound is purely inspiratory) in contra-distinction to *thymic stridor*, which may resemble it clinically, but differs from it essentially in its nature.

Opinions are still divided in regard to the changes in the larynx which are responsible for the symptom of stridor. Some authors (Thomson, Stamm, Turner, &c.) assume that there is some nerve disturbance, dependent upon an interference with the development of the cortical centres for the larynx, resulting in a disturbance of co-ordination in the respiratory act. Other authors assume that there is some arrest in the development in the region of the centre for the recurrent nerve, interfering with the function of the muscles of the vocal cords, the abductors of the glottis being more affected than the adductors (Kulner). Fullerton assigns the cause of the stridor to some form of unusually severe irritation, or to a special sensitiveness of the nerve endings with which the adductors of the glottis are provided. Smith assumes that the stridor is a reflex from adenoid growths which cause spasm of the aryepiglottic folds, an assumption which is untenable, because newborn infants do not suffer from adenoids.

In Truapp's opinion, a congenital weakness of the musculature of the glottis is the cause. He bases his view on the researches of other authors, who attribute the threatening symptoms which occur in diplophonic stenosis of the larynx in infants to the feeble development of the muscles of the glottis—especially the posterior crico-arytenoids—in early infancy. According to this view, the sound originates in the rima glottidis.

The prevalent view is, however, that the cause of congenital stridor is to be found in peculiarities in the entrance to the larynx. In infants the larynx is softer and more flexible, and its entrance is narrower than in older children. Peculiar alterations in the shape of the larynx in infants with congenital stridor are seen at autopsies, and are also visible on direct inspection with the autoscope. These abnormalities consist of narrowing and a groove-like deepening of the epiglottis with an in-folding of its edges, great approximation

of the arytenoid cartilages, and an abnormal length of the ary-epiglottic folds (Reardon). These changes may determine a considerable narrowing of the laryngeal orifice, especially during inspiration, when the flexible parts are all exposed to the suction action of the current of air (Finner, Thomson and Turner, Variot, Ballin, Finkelstein, Bokay, &c.).

It is not yet decided whether these alterations in the shape of the larynx are to be regarded as congenital maldevelopments, or whether they are secondary changes. According to Ballin, the larynx in children with stridor is definitely smaller than in normal children, notwithstanding that comparative sections made through the larynx have shown no differences, as far as the muscles and cartilages are concerned. It is possible that the pronounced alterations in shape only develop fully after breathing is established, on the basis of the congenital softness and flexibility of the part.

The mechanism which causes the moanlike sound is the folding together of the laryngeal orifice, so that a pipeslike structure is formed. Paterson thinks that the tone is due to the vibration of the soft parts on the posterior wall of the larynx.

No treatment is required in uncomplicated cases. There is rational foundation for the use of calcium chloride, as recommended by Neuter, in congenital stridor, in view of its etiology. There is hardly ever any indication for intubation, which, according to Variot and Trumpp, causes a cessation of the stridor. The same remark applies to excision of the ary-epiglottic folds, as suggested by Reardon. From the prophylactic point of view, it is essential to guard children with stridor most carefully from respiratory diseases. An ordinary laryngeal catarrh is apt to provoke symptoms of suffocation, and the slightest sign of such a condition demands prompt counter-irritant measures to the skin (Ballin).

(b) THYMIC STRIDOR.

Ayell first advocated the view, which was subsequently emphasized by Hochsinger, that the stridulous sound in congenital stridor was always due to compression by a hypertrophied thymus gland. They accordingly proposed to substitute the term thymic stridor for congenital stridor. This view is not generally held at the present day, although it is agreed that there is a form of hypertrophy of the thymus which gives rise to symptoms of obstruction in young children. These symptoms may possibly differ from those of congenital laryngeal stridor. Hochsinger defines his "congenital or thymic stridor" as follows: "A noisy respiration which is congenital or comes on within the first few months; its intensity is most marked at the end of inspiration, it persists by day and by night; every breath is accompanied by a rattling, bleating or clucking sound. Supra- and sub-sternal inspiratory retraction is constant, showing that there is some obstruction in the upper air-way." Hochsinger states that the symptoms do not

usually appear at birth, but come on after a few months. Accordingly, thymic stridor does not really come within the category of diseases of the newborn; at any rate the justification for the term "stridor neonatorum" may well be questioned.

Skodowsky advances the objection that stridor due to the thymus cannot be inspiratory in character. He remarks that thymic asthma must be expiratory, and must necessarily resemble the dyspnoea caused by mediastinal tumours, which compress the trachea and bronchi in the expiratory phase. The thymus gland, which lies between the sternum and trachea, is compressed during expiration, and it presses against the neighbouring organs; but it is turned away from the trachea during inspiration, when the capacity of the chest is increased. If an enlarged thymus causes stridor during inspiration, this symptom must be worse during expiration, because this is the phase wherein the gland is deflected from its position, e.g., towards the jugular fossa. As most cases of congenital stridor do not manifest any symptoms of expiratory obstruction, it is quite clear from the clinical standpoint that there is no basis for the view that enlargement of the thymus is the principal or even the only cause.

The following facts are brought forward as proving the existence of thymogenous stenosis of the trachea.

(1) Signs of tracheal flattening as a result of enlarged thymus. This appearance which was found especially by Flügge and Hedinger in cases of sudden death in young children, does not finally decide this question, and for two reasons. On the one hand cases of stridor are reported with enlargement of the thymus which led to sudden death, whilst no appearances of compression in the trachea could be proved (Probsting); on the other hand Richter considers that the flattening of the trachea, observed in hardened preparations, is an artefact.

(2) Proof of enlargement of the thymus gland at the post-mortems of children having succumbed to symptoms of stenosis. Considerable caution should be shown in deciding whether the gland is enlarged from its weight and measurements as estimated at the autopsy. According to Richter, in the newly born thymus glands of 20 to 24 gm. are not infrequent, and the case described by Perrin de la Touche of a thymus weighing 17 gm. falls within the normal. Skodowski examined the thymus of 150 newborn bodies, and found in twenty-one cases a weight of 16 to 27 gm.; no symptoms of asthma or thymus death having been observed in any of these cases. The length measurements also under normal conditions seem to fluctuate considerably. Concerning the effect of pressure, the absolute size seems to be of less importance than the thickness in diameter (from the front to the back) and the consistency of the organ.

Undoubtedly there are pathologically enlarged thymus glands. Klose observed a case in which the thymus filled the greater part of both thoracic cavities (four hours after birth), and Rubsamen

observed a child born by facial presentation with an enormous "goitre," mostly formed by hypertrophy of the thymus. Large thymus tumours have been repeatedly observed in children with malformations (Henning, Hedinger).

(3) The proof of an enlarged thymus in the Röntgen picture (fig. 59). It was chiefly this symptom which gave rise to Hochsinger's theory of the thymogenous nature of stridor congenitus. According to Hochsinger, there is a typical Röntgen picture of the thymus which is depicted as a concave band, parallel from the shadow of the heart with the shadow of the vertebral column.

Under normal conditions the width of this band at the level of the attachment of the second rib to the dorsal vertebra is only slightly broader than the shadow of the vertebral column. Under pathological conditions the part of the thymus shadow lying over the shadow of the vertebral column becomes broader, so that on both sides it extends beyond it, and apparently enlarges the shadow



FIG. 59.—Widening of the upper portion of the central thoracic shadow on skingram, in a child five weeks old, with congenital stridor. (Case of E. Baer.)

of the heart sideways. By means of the Röntgen method Hochsinger was able to establish thymus hypertrophy in infants with stridor congenitus. Other authorities, however, were not able to confirm this condition. Skolow saw two shadows passing over the margin of sternum, but considers it difficult to decide whether they referred to the thymus, the blood-vessels, or lymphatic glands. According to Benjamin and Gint, the Hochsinger "thymus shadow" is also found in perfectly healthy children, and appears to originate not from the thymus, but from the superior vena cava. In the same child the shadow may appear in the shortest time, quickly disappear, exhibit a displacement simultaneously with respirations, inasmuch as with inspiration it rises well into the right pulmonary region, and disappear with expiration to the margin of the vertebral column. In consideration of these observations not too much importance must be attached to the symptom, observed by Rein, of enlarged thymus, consisting of diminution of the shadow with

inspiration and increase with expiration, and considerable caution should be exercised in respect of the Röntgen examination.

(4) Enlargement of thymus can be but seldom proved convincingly by percussion. Blumenreich's and Friedjung's area of dullness of the thymus presents an equilateral triangle, whose base is formed by a line between the sternoclavicular joints and whose rounded apex is situated at the level of the second rib or a little lower. Great precaution must therefore be exercised concerning the value of results, and in particular one should be most cautious in one's judgment regarding the diagnosis of an enlarged thymus with only slight dullness of thymus. The more over the manubrium sterni and the dullness caused by neighbouring organs, in particular the heart, must be taken into consideration. Only very pronounced dullness at the characteristic spot should be attributed with any probability to the thymus, and such is extremely rare. It must also be considered that for the occurrence of compression of the trachea an enlarged thymus proved radiologically or by percussion is not necessary, for as already mentioned the size of the organ is not the decisive factor.

(5) Palpation of the thymus gland has succeeded in some cases, as with expiration a soft tumour has been felt in the jugular fossa. With simultaneously existing symptoms of stenosis this symptom, if pronounced, is diagnostically of value.

(6) Though many of these arguments concerning the occurrence of thymogenous stridors have been contested, the participation of the thymus in the existence of certain symptoms of stenosis has been proved by the results of operations. Kluse and Ssokolow have collected the cases hitherto operated. The following cases concerning the newly born period have been taken from their table:—

(1) König's case. (Age at operation 9 weeks.) Attacks of asthma and cyanosis since the second week. After tracheotomy attacks of asthma with severe inspiratory and expiratory restriction continued. A soft tumour in front of the trachea. On depression of this mass, which proved to be the thymus, relief in breathing. A portion of the thymus was resected, and the rest sown freely to the manubrium sterni with output. Recovery.

(2) Penzler's case. (Age at operation 2½ years.) Since birth, loud long-drawn respiration, hoarseness. Increase of obstruction in respiration, so that tracheotomy had to be performed. The enlarged thymus was seen with inspiration in the form of a white mass. The gland was entirely enucleated, and the stridor immediately disappeared. Recovery.

(3) Rehn's case. (Age at operation 4 months.) Difficulty in breathing since birth, increasing with excitement. Attacks of cyanosis. Inspiration difficult; expiration easier but accompanied by a forward bulging above the manubrium sterni. *Operation.* Skin incision, blunt separation of muscles, incision of deep fascia. On inspiration, the thymus bulged forward into the wound; on expiration it retracted. The gland was ligated on being seized by forceps. Its capsule was adherent to the fascia above the manubrium sterni. After the operation cyanosis disappeared and easy breathing was established.

(4) König's case. (Age at operation 4 months.) Difficulty in breathing since birth. On expiration the thymus bulged and could be felt at the root of the neck. Resection of left lobe of thymus, and then slitting of the stump. As the breathing was not free, tracheotomy was performed. At a second operation the remains of the left lobe were removed. After resection of the upper border of the sternum the right lobe was drawn forward, then, at last, breathing became free.

13) Schottin's case. (Age at operation 25 days.) Difficult breathing since birth. Loud stridor on inspiration and expiration. During expiration a small swelling seemed to be jerked forward at the root of the neck. Partial resection of thymus gland. Complete recovery.

14) Mott-Murphy's case. (Age at operation 11 weeks.) Extreme difficulty in breathing since birth. No swelling could be detected either on palpation or on percussion. After resection of thymus gland adhesion of capsule of thymus was evident. The respiratory symptoms improved, but the child died in sixteen days.

15) Vein's case. (Age at operation 11 months.) Since birth severe suffocative attacks and loud stridor. Intracapsular excision of thymus. After the operation the apnoeal attacks ceased, but the stridor continued.

The results of operative removal of the whole or part of the thymus gland afford the clearest proof of the actual existence of a thymic asthma, of a tracheal obstruction due to the pressure of a hypertrophied thymus. The symptom is not by any means peculiar to newborn infants, but whenever the symptom is present it usually dates back to early infancy. The diagnosis is difficult, and one can only be sure about it in very pronounced cases. Klose states that "the diagnosis of thymus compression in cases in which physical signs are absent can be based on the presence of chronic signs of obstruction of the organs situated deeply in the neck, associated with acute paroxysmal exacerbations, or on the occurrence of dangerous attacks of dyspnoea with the appearance of a swelling of the root of the neck during expiration." But there are undoubtedly milder cases in which there are no attacks of dyspnoea, and in which a swelling does not appear. The mild cases of thymic stridor should improve as the child grows older, because the trachea becomes more rigid. During the first days of life an enlargement of the thymus due to hyperaemia may be a serious matter, just as happens in the case of swelling of the thyroid gland. It is quite possible that there may be a thymic stridor with a maximum intensity in the first days of life; but its existence has not yet been proved.

The situation of the tumour within the thorax is the cause of the type of dyspnoea and stridor being mainly expiratory, hence the term thymic "asthma." There are cases in which the peculiar stridulous breathing and the dyspnoea are both absent and the only signs of compression by the thymus consist in the audible expiration and the somewhat increased frequency of breathing (Unger).

The dangerous symptoms which may befall infants with thymic stridor demand treatment. As long as the signs of stenosis are not severe, one may obviously temporize, because as the child grows the thymus becomes smaller and the trachea becomes more rigid. So far the prognosis is favourable. But if the stenosis really prevents the access of air to the lungs, we cannot secure an entry of air by the usual methods of intubation or tracheostomy, because the obstruction is too deep-seated. Theoretically, the adoption of tracheotomy appears rational, because a catheter can be introduced through the wound; but this operation is so liable to be followed by pneumonia in the newborn that it becomes a very dangerous procedure. The most practical operation is intracapsular

dislocation of the gland and ectopexy; if this does not suffice, the gland must be partially enucleated or excised. Finally, the operation of partial removal with re-fixation after resection of the manubrium sterni may be carried out (Klose and Vogt). The cases previously detailed show that operative interference should not be undertaken until the infant is over a month old.

Thymic Death.

It is now necessary to refer to a form of sudden death, which is described as of frequent occurrence in infants, and which is designated by the term "thymic death." This term does not indicate a mode of death by suffocation, coming on after symptoms of obstruction, it is rather a totally unexpected death by asphyxia which occurs in infants immediately after birth or a few hours later, more rarely during the first few days. In the latter case, the children may have already manifested some stridor, and death occurs during a sudden exuberation (Summa); or the children may apparently be quite healthy and show no signs of respiratory embarrassment.

There are two explanations offered to account for the rôle of the thymus in the causation of sudden death. The gland possibly presses upon the trachea and against the large vessels and thus mechanically causes suffocation and death; or death may be the result of *ataxia thymicodysphagica*, an anomaly of the constitution which is held responsible for sudden and unexpected deaths of apparently healthy individuals later in life.

Most authorities consider that thymic death in infants is to be explained on the mechanical theory. If the infant dies shortly after birth, before complete recovery from the asphyxia of the labour, we cannot assume that the thymus was the one and only cause, unless there is anatomical proof of the enlargement of the gland and of its pressure upon the trachea and large blood vessels. Such cases have actually been described by Flügge, Summa and Heding. The last named author reports seventeen cases of apparently healthy full-time or premature infants who died very shortly after birth, with signs of asphyxia; in all of these, autopsy revealed a more or less pronounced hypertrophy of the thymus, occasionally associated with enlargement of the thyroid gland.

In order to explain sudden death in apparently healthy infants, a few days old, it is necessary to assume a sudden hyperæmic swelling of the thymus. According to Klose, this is quite a feasible contingency. He points to similar conditions in intra-thoracic gaiter, wherein surgical experience shows that suffocation or signs thereof may take place quite suddenly in the midst of apparent health. Baginsky found that the thymus gland completely surrounded the trachea of a child who had died suddenly, thus showing that death was due to suffocation.

In some cases, severe hæmorrhage would appear to be the cause of the sudden death, i.e., apoplexy of the thymus gland (Baeren-

spring, Raudnitz, Schlesinger, Mendelssohn), but these cases are exceedingly rare. In those hitherto recorded there was a syphilitic basis for the hæmorrhage in some and in others the hæmorrhage occurred into a pre-existing cyst.

The cases hitherto observed do not show any clear relationship to status lymphaticus as seen in older children. Penkert, however, reports two cases of sudden death immediately after birth wherein enlargement of the mediastinal glands was discovered (thymic death), and Unger reports a case of enlargement of the thymus demonstrated by percussion and X-ray examination, wherein there was hypertrophy of almost all the lymphatic glands that could be felt, and of the spleen. This is not the usual condition. Hedinger's specimens showed no hypertrophy in the lymphatic system, accompanying the enlargement of the thymus, and the chromaffine system was normal to the naked eye and also on microscopic examination. Lohrsech was unable to find evidence of status lymphaticus in his examination of fetuses, newborn infants, or those who died within the first two weeks. Nevertheless some disturbance in the secretion of the thymus may exist and play its part in the causation of sudden death, but there is no definite support for this assumption.

Some doubt may well be entertained in regard to the causal relationship of sudden death to the thymus gland. Cases of sudden death occur wherein the post-mortem fails to reveal the slightest enlargement of the thymus. And even if we do find a large thymus and signs of compression of the trachea we must remember that these symptoms are somewhat unreliable and the actual cause of death may be something quite different altogether. Skokolow has collected twenty-nine cases of sudden thymic death in newborn infants. The critical examination of these cases seems to justify the conclusion that death was really due to injury during labour and the consequent asphyxia. We can only take it as proved that the thymus may produce signs of obstruction and may be the cause of death in a case presenting such symptoms. There is, however, no scientific basis for asserting that the thymus is the cause of those sudden deaths by asphyxia which occur immediately after birth or within the first day or two of life.

(c) CONGENITAL STRIDOR ARISING FROM OTHER CAUSES.

We are now about to discuss certain diseases of the newborn, which are accompanied by more or less pronounced stridor and which are allied clinically with the conditions described above.

The cause of an inspiratory stridor may be situated above the larynx, e.g., in the *nostrils*. Congenital tumours in the region of the floor of the tongue and entrance to the larynx (cyst of the thyro-glossal duct, dermoids) may cause harmless symptoms of stridor, but they may also cause severe dyspnoea and even dangerous signs of asphyxia (Finkelstein). Shukowski has described two

cases of small-jawed infants, wherein the tongue, which had fallen back, caused symptoms of stridor (whistling inspiration) and respiratory embarrassment accompanied by cyanosis. He recommends that the tongue should be drawn forwards in these cases, and sutured to the lower lip or cheek. Seokolow also refers to the fact that stridor can be caused by the falling back of the tongue, which need not necessarily be enlarged, but may indeed be abnormally small.

Hohlfeld has described a case wherein inspiratory stridor with attacks of suffocation had existed since the first weeks. There was also a difficulty in feeding the infant. The child died at the age of 3 months, in a severe attack of asphyxia, for which tracheotomy was performed. The post-mortem revealed a tumour (*leiomyoma fibrosum*) on the posterior wall of the larynx, where the pharynx opens into the oesophagus. The tumour had been compressing the larynx and the trachea as far as its bifurcation.

Congenital stenosis of the trachea usually leads to rapid death, but, exceptionally, life may be prolonged for some time (Gregor). Mimosons reports the case of a child which had suffered continuously since birth with severe dyspnoea and cyanosis, although the voice was clear. Death occurred in the third month, and the post-mortem revealed stenosis and torsion of the trachea below the cricoid cartilage. O'Dwyer observed a case of *congenital maldevelopment of the larynx*, consisting of a fusion of the ary-epiglottic folds and narrowing of the entrance to the larynx. This caused dyspnoea, which had existed since birth. A cure was effected by means of gradual dilatation with urethral bougies. Rothschild has reported a case of "typical congenital stridor," caused by a submucous retention cyst arising from a mucous gland of the larynx. The cyst suppurated and caused death.

Congenital hypertrophy of the thyroid gland is a comparatively frequent cause of respiratory embarrassment. The relatively small *retrosternal goitres* may lead to difficulty in diagnosis, because they may produce dangerous asphyxia and cyanosis, without any clinical evidence of obstruction (Bercow).

Inflammatory diseases of the pharynx (retro-pharyngeal lymphadenitis and abscess) and of the larynx (croup, false croup) which are frequent in young children and are accompanied by stridor, are very rare in newborn infants. But if an inflammatory swelling of the laryngeal or tracheal mucous membrane does actually occur, dangerous symptoms are very apt to develop, because the lumen of the windpipe is easily obstructed, especially if there is considerable secretion. A tracheitis accompanied by great swelling of the mucous membrane may demand the performance of intubation, even if the larynx remain free. The infants are usually 2 to 3 weeks old when these symptoms appear. If a child who suffers from congenital stridor become affected by laryngitis, symptoms of false croup are easily provoked (Finkelsarain). Shulzowski has described a case of ulcerative laryngitis, possibly of syphilitic origin, which was the cause of a stridor, persisting since the second day of life.

Pressure of a congenitally mal-developed heart upon the recurrent laryngeal nerve may cause paralytic symptoms of that nerve (Hauser, Hochsinger and Finkelstein). These symptoms consist of hoarseness, and persistent or paroxysmal disturbances of breathing (stridor). Newborn infants do not suffer from true laryngeal spasm (*vide infra*).

The following case of the author's will indicate how difficult it may be to establish the cause of congenital stridor—even at the autopsy. An infant, born after a normal labour, manifested from the moment of birth a marked inspiratory and expiratory stridor, which increased from hour to hour and which was accompanied by dangerous symptoms of suffocation. Intubation was not successful, though the skiagram showed that the tube reached down to the root of the neck. As it was thus possible to exclude an obstruction in the larynx or from pressure of the thyroid gland, the thymus was removed twelve hours after birth. But the symptoms did not improve, and the infant died one and a half hour after the operation. The post-mortem showed that only the right lobe of the thymus had been removed; this weighed 6 grammes, the rest weighed 4 grammes. The anatomical cause of the symptoms of stenosis could not be demonstrated with any certainty; but there was some slight kinking of the tracheal cartilages at the level of the thymus. Nevertheless the stridor may have been of thymus origin, although the gland was not in any way abnormally large, and a large shadow seen on the skiagram, before the operation, was certainly not cast by the thymus.

(III) Diseases of the Bronchi and Lungs.

(1) Congenital Anomalies.

FETAL BRONCHIECTASIS.

The so-called congenital bronchiectasis (Grawitz) comprises two forms; the generalized form wherein the condition affects an entire bronchus by reason of oedematous degeneration, and the telangiectatic form wherein the bronchi develop cystic dilatations, either as single cysts or multilocular spaces (cystic lung, honeycomb lung) (Convalaire, Peiser, Stoerk, &c.).

In other cases, the condition is related to foetal atelectasis, when it is called atelectatic bronchiectasis. Atelectasis is caused by the failure of portions of the lung to expand, having become compressed and fibrotic by the growth of the bronchial ramifications, or it may be the result of a foetal pleurisy which also restricts the expansion of the lung tissue. On the other hand, some look upon atelectasis as a simple delay in development (Buchmann). Peiser regards it as secondary in congenital bronchiectasis.

The clinical signs of foetal bronchiectasis are not very characteristic. If the atelectasis or the cystic changes are very extensive,

the infants die soon after birth, with signs of asphyxia. If the changes are slight, there may be no symptoms at first. Later on, inflammatory complications supervene, and the symptom-complex of bronchiectasis appears.

MALFORMATION (HYPOPLASIA AND HYPERPLASIA) OF ONE LUNG.

The lungs may be malformed, either through defective or excessive development. In hypoplasia (agenesis) a small airless structure may be found in the place of the lung. As the healthy lung usually extends into the empty side of the chest, and as the thorax on the sound side does not develop normally, no thoracic deformity usually occurs (Ponfick, Neisser, Oberwarth). The same applies to cases of excessive development. There may be primary hypertrophy of one lung (Griff) or there may be normal development of both lungs, but in addition thereto, small structures of lung tissue, forming a third odd lobe.

If such malformations are at all compatible with life, the patients, so affected, may reach old age. Maldevelopment is sometimes associated with bronchiectasis.

Owing to the compensatory growth of the other lung, the diagnoses can only be made during life, if there be dullness over one half of the thorax. Such dullness in older children would, however, always be attributed to infiltration or exudation. In newborn infants the true condition might be suspected, but as a matter of fact the diagnosis has hitherto not been made.

(2) Atelectasis of the Lung.

The lung does not undergo complete expansion at the first breath. The researches of Dobson and Recklinghausen show that the respiratory exchange is extremely small on the first day.

Measurements of the lung capacity in the first few hours of life yielded the following results:—

| | |
|--------------------------------------|---------|
| 20 minutes after birth | 17 c.c. |
| $3\frac{1}{2}$ hours after birth | 21 c.c. |
| Within the first 6 hours after birth | 36 c.c. |

The depth of inspiration acquires a considerable increase between the first and the second day, but many days pass before complete expansion of the lungs occurs. Peser attributes this circumstance to the incomplete development of the nervous system, to the relatively slight reaction of the respiratory centre to the irritation of CO_2 , and to the fact that infants move very little and therefore the muscular system only produces in very small amount those irritant substances which stimulate the respiratory centre. The expansion of the lung is longer delayed in debilitated and premature infants. During the first day, the expansion of the chest is mainly due to the diaphragmatic movement, but later thoracic breathing becomes gradually established.

It follows, from the above, that atelectasis is to a certain extent, a physiological—though temporary—state in the first days of life. The transition to the pathological state is quite rapid.

Congenital atelectasis is either general or partial. The former occurs in infants who have never breathed, and therefore has only an anatomical interest. Peiser has shown, by means of sections of the thoracic viscera fixed in situ, that the most extensive atelectatic areas occur in the paravertebral (especially in the upper part) and central portions of the lung, at the hilus; whereas the apices and the borders rapidly become aerated. The left lung is usually more atelectatic than the right.

Atelectasis is frequently complicated by hæmorrhages and œdema. The cardiac action is weak owing to the defective exchange of gases. Stasis occurs, to be followed by the exudation of blood and serum from the capillaries, whose walls have become damaged by the stasis. Hæmorrhages occur mostly in the vicinity of the hilus. If the damage to the capillary walls is but slight, nothing occurs beyond œdematous exudation.

Those portions of atelectatic lungs which are infiltrated with blood or tissue fluids are favourite sites for the development of pneumonia.

Pathological atelectasis and its sequelæ are especially frequent in premature and debilitated infants, and in those who have suffered from asphyxia during birth. In the former type the excitability of the respiratory centre is *a priori* slight, and in the latter type its excitability has been reduced by the asphyxia. Damage to the respiratory centre may also result from increased intra-cranial pressure. Effusion of blood into the cranial cavity has often been observed to be followed by extensive pulmonary atelectasis (Kundrat). The respiratory excursions are not great enough to expand the lungs in full measure, and therefore many parts remain atelectatic. The exchange of gases is but slight. The infants, who are born asphyxiated, become cyanotic again after their resuscitation; they do not really emerge from the state of asphyxia. Others lie in a sleepy condition, with scarcely perceptible respiratory movements, and then suddenly become cyanotic. The breathing becomes more and more unsatisfactory, and finally absolute pauses occur therein—in short, a picture develops which is analogous to the state of apparent death, sometimes seen in infants just delivered. Life soon ebbs away completely in these cases.

If the respiratory excursions are extensive enough to ensure an adequate supply of oxygen, inflammatory complications constitute the chief danger to life. Pneumonia, which is very apt to supervene when the risk of infection exists, in the atelectatic portions of the lung, is a very frequent cause of death in premature infants.

The diagnosis of atelectasis can be based more definitely upon the clinical picture of shallow respiration and cyanotic aspect than upon physical signs. It is very difficult to percuss out areas of

dullness unless the lung is extensively consolidated. The most striking physical sign is the so-called atelectatic crackling, which is occasionally heard in a very pronounced unmistakable manner after inducing the infant to take a deep breath by means of a slip. Peiser holds that this indicates the passage of fluid into the alveoli. The other signs of infiltration (bronchial breathing, bronchophony) are not usually very distinct, because the respiratory excursions are too defective.

The treatment of atelectasis must be directed towards expanding the lungs, by stimulating deep respirations. In mild cases it will suffice to apply such cutaneous stimulation as will cause the infant to breathe deeply and to cry. These infants must never be allowed to lie quietly on their backs for any length of time. The most powerful stimulus is effected by cold affusions while the infant is in a hot bath or a mustard or oxygen bath. The bath produces vascular dilatation in the skin, and thus prevents over-filling of the lungs with blood. If an attack of asphyxia comes on, it is necessary to employ all the methods which are used in the case of stillborn infants.

In addition to fetal or congenital atelectasis, infants may also suffer from acquired atelectasis, which occurs in two forms—compression atelectasis and obstruction atelectasis. The latter variety is more frequent in newborn infants. An attack of bronchitis may cause the blocking of a bronchus, and this may easily lead to atelectasis, especially if the bronchus supplies a section of lung which is inadequately ventilated. In such a case the transition to inflammatory infiltration is particularly easy.

(3) Hypostatic Congestion of the Lung.

Symptoms of congestion, accompanied by effusion of blood into the alveoli, may occur in the posterior and inferior portions of the lungs in cases of congenital defects of the heart. If the congestion is severe, it is liable to be followed by exudation and comparative induration of the interstitial connective tissue of the lung. The clinical symptoms consist of respiratory embarrassment; at first the breathing is superficial and irregular, but later on attacks of asphyxia occur. There is also dullness on percussion, which may be confused with pneumonia or atelectasis (Hayashi).

(4) Inflammatory Diseases of the Lungs and Brouchi.

Inflammation of the lungs may arise from infection through the following paths:

- (a) Intra-uterine infection.
- (b) Infection during labour (aspiration of liquor amni or vaginal secretion containing microorganisms).
- (c) Extra-uterine infection (air-borne infection, aspiration of milk, vomit, &c.).

CONGENITAL PNEUMONIA.

If the infant of a mother who is suffering from sepsis contracts pneumonia within the first day or two of life, it must not be assumed forthwith that the infection travelled by the placental route. The infection may have been derived from the amniotic fluid (*vide infra*), or may have occurred intra-partum. It is not always easy to exclude, with certainty, a pneumonia due to the aspiration or inhalation of infective material post-partum. It requires careful consideration to establish the date of infection. Finkelstein suggests, for instance, that influenza may cause widespread infection within a few hours.

The number of cases of congenital pneumonia, wherein there is justification for assuming that the infection has been conveyed through the placenta, is quite small (*vide infra*).

Levy reports the following case: A mother had been suffering from pleuro-pneumonia for some days before and also during her labour. The infant, seven hours after its birth, showed signs of cyanosis, dyspnoea, and fine crepitant râles, and only lived for forty-nine hours. The autopsy revealed a hæmorrhagic and catarrhal pneumonia of the right side, which Recklinghausen considered was at most of three days' duration. The case was regarded as a genuine croupous pneumonia, acquired *in utero*, also the microscopical examination of the placenta proved to be negative. The *Diplococcus pneumoniae* was demonstrated in the lungs of mother and child.

Thorner also reports a case wherein the mother had pneumonia, and the infant died two days after birth from lobar pneumonia. Macdonald reports another wherein the mother had pneumonia, and the infant died in twenty-eight hours from a solid pneumonia in the stage of red hepatization.

A case described by Bochenksky-Groebel is very convincing. A mother was suffering from pneumonia of the right lower lobe. Her infant was cyanotic and dyspnoeic immediately after birth; extensive dullness and numerous crepitations were detected in the chest, and the breathing was hurried. The mother recovered, but the infant died eleven hours after birth. The autopsy showed that all the lobes, with the exception of the apices, were affected by hæmorrhagic lobular pneumonia and by bronchitis (*Diplococcus pneumoniae*). The brief survival of the infant and the physical signs found immediately after birth render it very unlikely that the infection could have been post-partum.

We must assume that in these rare cases the infection came *via* the blood, and was conveyed by the migration of bacteria through a placenta which was probably damaged by toxins. It is remarkable that the infants did not always suffer from a generalized pneumococcal septicaemia, but that the pneumococci merely infected the foetal lung. Niederhof, who described a case of extensive congenital streptococcal pneumonia, assumes that there was an intra-uterine infection owing to the aspiration of liquor *amni* containing cocci. This mode of infection certainly explains a generalized

pneumonic process, but it does not meet the cases wherein the disease is limited to one lung or one lobe. In these cases we must assume that there is a special affinity for the lung on the part of the pneumococcus which has penetrated into the blood-stream.

ACQUIRED DISEASES OF THE LOWER AIR-PASSAGES (EXTRA-UTERINE).

Bronchitis or pneumonia acquired during birth or immediately after are due to aspiration or inhalation of infective material, or they may be symptoms of a general septic infection (Küstner, Geyl, Netter).

There is always serious danger of the development of an inhalation pneumonia, when the liquor amnii is infected or the vaginal secretion is septic. The researches of Hochheim show that infants very frequently make movements of inhalation, although they may be very slight, for he found some cells of pavement epithelium or droplets of fat in the lungs of nearly all the cases he examined. As the vaginal secretion nearly always contains micro-organisms, every deep inhalation provides the opportunity of inflammatory disease of the air-passages.

The lungs may be the portal of entry for the infection which causes septicaemia. In other cases pneumonic or haemorrhagic foci occur in the lungs as sequelae of a pre-existing sepsis. These may be caused by an embolic process, e.g., metastatic pneumonia from umbilical endometritis (Wassermann), or they may result from a secondary infection of the lower air-passages, which is very liable to occur in those infants who have lost their powers of resistance owing to their generalized disease.

The prognosis of pneumonia which comes on within the first day or two, and which usually runs its course with terrible rapidity, is absolutely hopeless. It is a little more favourable in cases where the process extends downwards gradually after air-borne infection, as in bronchitis and broncho-pneumonic foci following diseases of the upper air-passages. The result depends upon the virulence of the infective organisms (streptococcal processes are usually more dangerous than pneumococcal), or the extent of the disease (involvement of one or two lobes, or general extension; capillary bronchitis), and finally upon the general vigour of the child.

Two circumstances favour the onset of inflammatory processes in the lungs. As already stated, atelectatic portions of the lung form admirable soil for pneumonic foci. The aspiration of fluid is another important factor in weakly and unconscious children. These children are often quite unable to suck, either from the breast or the bottle. If spoon-feeding elicits a deglutition reflex this method of nutrition is certainly the proper one. But if this is unsuccessful, or only partially successful, some of the milk or other fluid trickles into the trachea, and may be aspirated into the bronchi, owing to the absence or inadequacy of the reflex irritability.

The fluid, by mere mechanical action, alters the mucous membrane and renders it a good soil for infection, or it sweeps into the lower air-passages some of the infective material which is always present in the bacteria-laden oral cavity. In this way danger may lurk in measures actually taken for the infant's benefit. When it is merely a case of "swallowing the wrong way," the danger of inhalation can be avoided by feeding through a tube. But in many cases it is not the food given by the mouth which is inhaled, but the contents of the stomach which are regurgitated or vomited. Pneumonia in atelectasis, as well as inhalation bronchitis or pneumonia, is a frequent terminal symptom, and often the direct cause of death in premature and debilitated infants, and in all conditions which are associated with a loss of consciousness.

The diagnosis of bronchitis and pneumonia is very difficult in young infants. The physical signs of bronchitis (dry and moist râles) and of infiltration (increased resistance, dullness, bronchial or high-pitched breathing, bronchophony, consonanting râles) are often very indefinite or entirely absent, especially if the diseased area is not very large. The absence of symptoms is due to the slight amplitude of the respiratory excursions, and also to the fact that the atelectatic area is in the central portion of the lung where no respiration occurs at all. If no air enters the diseased area and its vicinity, we cannot expect any definite auscultatory signs. It is necessary to make the child breathe deeply or cry in order to hear anything abnormal. Percussion is of value only if the exudation is extensive. If the area is small, the elasticity of the infantile thorax prevents any conclusion being drawn from physical signs, especially if the exudation is central. Even if there are definite signs of crepitations or dullness to establish a diagnosis, the autopsy generally shows that the lesion is much more extensive than the physical signs would indicate. The pulmonary complications are therefore a matter of inference from the general clinical picture, rather than from the signs derived from physical examination. Increased respiratory frequency, the movements of the rib cage, marks of cyanosis constitute valuable indications; but not much significance is to be attached to the temperature.

(5) Emphysema.

Emphysema of the lungs only occurs spontaneously in newborn infants in the form of acute distension of the lungs; this is a very infrequent condition, because the morbid states which lead to emphysema and to the forced respiratory movements which cause distension of the lungs are comparatively rare. Esser points out that disturbances in the innervation of the respiratory centre (e.g., powerful vagus stimulation) may result in distension of the lungs, and in these cases secondary hemorrhages are liable to occur owing to sudden over-stretching of the pulmonary vessels.

Kirchgeßner reports a case of *subcutaneous emphysema* in an asphyxiated child who had presumably inhaled a plug of mucus

during intra-uterine breathing. Lateral compression of the thorax produced a rupture in the affected lung owing to excessive pressure, and this led to interstitial and subcutaneous emphysema. The prognosis of this form of emphysema is quite favourable, as it may abate within a few days (Wilkins).

The attempted resuscitation of stillborn infants by pumping air into the lungs may produce emphysema, as the result of traumatism.

Injuries of the lung, or of the costal or visceral pleura by a fractured clavicle, may admit air into the pleural cavity or mediastinal spaces, and in this way may lead to pneumothorax with interstitial, mediastinal and subcutaneous emphysema. It is quite likely that Schulze's manoeuvres for the resuscitation of infants may also be responsible for some cases of emphysema (Heydrich).

(IV) Pleurisy.

Pleural effusion is very rare in newborn infants. Serous pleurisy of tuberculous or rheumatic origin has hardly ever been observed. Fibrinous, fibrino-purulent and entirely purulent effusions occur as complications of pneumonia, especially of the septic variety. These forms of pleurisy are usually manifestations of a generalized infection, of a pyæmia, arising from primary infection of the lungs, or from some other source (Steele). Empyema occurring in newborn infants is generally streptococcal (Gaye, Comby, Suhl, D'Astros). (*Vide infra*.)

The diagnosis is difficult because the physical signs are not often very characteristic. Comby considers the following to be the most important physical signs: Dry paroxysmal cough, high temperature of the remittent type, short jerky diaphragmatic breathing, with great dyspnoea and retraction, pale, cyanotic or jaundiced aspect, vomiting and great constitutional disturbance. But in the absence of definite dullness, it is not easy to diagnose pleurisy from these symptoms. If there be some œdema of the skin over the affected segment of the lung, it may be very suggestive for the diagnosis. The prognosis is so bad that even a well-established diagnosis hardly justifies a resection of the rib, because a fatal issue can hardly be prevented. The prognosis is somewhat better in an ordinary pneumococcal empyema and which may also occur within the first three weeks of life.

CHAPTER III

THE THYROID.

The thyroid gland is situated on a somewhat higher level in infancy than in later years. The extremities of the lateral processes usually project between the uppermost segment of the trachea and

oesophagus, or between the larynx and pharynx; more rarely between these and the spinal column. It follows therefore that enlargement of the gland may easily lead to circular constriction of the trachea (Demme). In some newborn infants it is quite easy to palpate the three lobes of the gland on dorsiflexion of the head. The size and weight of the thyroid vary considerably with the neighbourhood. In goitrous districts the glands are much larger than in non-goitrous districts, although they may not contain any nodules (Isenschmid). The average weight of a large number of thyroids belonging to fully developed fetuses, newborn infants and children up to the age of six months, was according to Hesselberg 1.33 gram. This applied to the districts of Kiel, Königsberg and Berlin, that is, the North German Landmarks. The average weight in Berne was 4.1 to 6.6 gram, although no obviously goitrous glands were included. Similar observations were made by Meroz-Tyldman in regard to the weight of thyroid glands in Geneva.

The most interesting of the abnormalities in position in which the thyroid is subject, is the one known as the retro-sternal. The gland is usually goitrous and its isthmus is deeply situated, or it sends a conical process under the sternum. Such retro-sternal goitres, which may easily lead to suffocation, occur very rarely.

E. Meyer has noted a case of an infant, hoarse since birth, who had a bluish-grey swelling resembling papilloma on the anterior commissure of the vocal cords. After its removal it was found to contain some thyroid gland tissue. This was evidently a congenital *endolaryngeal goitre*.

Inflammation of the thyroid gland may occur as a complication in septicæmic or pyæmic diseases, but it is a very rare occurrence.

Petravel examined an infant who died, when ten days old, from "purpura," and found peculiar areas of degeneration of the epithelial cells of the thyroid. He suggests that toxin in the blood produced this cell degeneration (Fitch, *Arch.*, 206, 1911, p. 1).

Hypoplasia or aplasia of the thyroid gland produces the signs of congenital myxœdema (v. p. 166). The pressure of tumours—especially teratomata—may result in partial or complete loss of the thyroid gland.

Graves' disease, which is caused by hyperactivity of the thyroid, is exceedingly rare in infancy. Clifford White reports as follows on a remarkable case of "congenital Graves' disease." The infant, whose mother had been suffering since the fifth month of pregnancy, from progressive symptoms of Graves' disease, had extreme exophthalmos, fine tremors of the hands, tachycardia with a pulse of 150-200 per minute, and also enlargement of the thyroid gland. Death occurred on the second day.

Congenital Enlargements of the Thyroid.

(1) Transitory Enlargements.

There is a type of swelling of the thyroid among newborn infants, which is characterized by its rapid subsidence—either after

a few days or within a few weeks. The gland is uniformly swollen throughout and its consistence fairly soft. The skin and subcutaneous tissue around may participate in the swelling. Sometimes, the swelling causes no symptoms; in others, the children are asphyxiated immediately after birth, or manifest temporary signs of suffocation during the first days, on drinking or extreme flexion of the head, or even in sleep. These symptoms do not as a rule become dangerous to life. The swelling subsides rapidly during the first days, and the symptoms of obstruction do not recur. This condition is sometimes associated with certain forms of congenital goitre with many serious symptoms—sometimes even fatal, as in a case recorded by Harker, but this is a great rarity. Probably these cases are combined with genuine goitre.

It must be assumed that the cause of transitory swelling of the thyroid is generally severe hyperæmia and oedematous infiltration of the gland tissue. The condition is sometimes called hyperæmic turgescence (Denne) or congestion of the thyroid gland (Fabre and Thérivent).

There are several factors to be considered in regard to the ætiology. Fabre and Thérivent assume that the congestion is favoured by the persistence of the fetal circulatory system and also by the persistence of the embryonal type of lacunar vascularization, as already described by Wüller. The most important factor would appear to be the excessive pressure during the process of birth. The frequent coincidence of goitre with face presentations shows that congestion plays an important part. It is obvious that a large swelling of the front of the neck will predispose to extension of the head with a resulting face presentation. On the other hand, a face presentation may be primary and the swelling of the thyroid secondary. This may occur when there is lordosis of the spine and the swelling must be regarded as a form of congestion produced by the pressure during parturition (v. Winckel, Kamann). This, however, is not the only cause, because congestion of the thyroid occurs also in cases of normal vertex presentations.

But there are other factors to take into account, besides congestion; these are either localized to the neck or are generalized. The turgescence of the thyroid may be regarded as analogous to the hyperæmic and oedematous conditions which Halbau looks upon as reactions of pregnancy (Lieber). In women the thyroid is definitely influenced by the sexual organs. The gland generally swells, not only during menstruation, but also during pregnancy and during parturition, and subsequently returns to its normal size (Freund). This swelling of the thyroid may be attributed to the effect of placental "pregnancy toxins," being one of the pregnancy reactions. The comparative rarity of this disorder in the infant is, however, an argument against this view, but it is quite possible that slight enlargements cannot be detected clinically or are overlooked. We have already stated that the thyroid is very often palpable in newborn infants. Whenever the enlargement is very obvious, the mother generally has greater swelling of the gland than is usual.

It is not always easy to differentiate this condition—at any rate on the first day of life—from a persistent goitre. The prognosis, of course, depends upon this differential diagnosis. The history may provide some indication. As the genuine congenital goitre is definitely hereditary, we may assume that the swelling in the infant is transitory, if the mother only acquired the swelling of her thyroid during pregnancy. On the other hand, a rapid subsidence of the swelling during the first days is no conclusive argument against a genuine goitre, because the latter may undergo transitory enlargement as a result of congestion.

(2) Permanent Enlargements of the Thyroid.

(1) CONGENITAL GOITRE.

Genuine congenital goitre is closely related to endemic goitre, and is definitely hereditary. Inquiry will usually show that an infant with goitre comes of a stock in which goitres have occurred previously, or comes of a family resident in a goitrous district. In most cases, the mother of the child is afflicted with goitre. The frequency of genuine congenital goitre varies in different neighbourhoods. In some districts it is quite rare; but observers who draw their material from Alpine regions or the mountains of central Germany (Thuringen) describe it as a comparatively common disease (Demme, Küstner, Rubsamen).

Endemic goitre usually occurs at the age of puberty or in the later years of childhood. The next period of frequency is between foetal life to the end of the first year. Demme found fifty-three cases of congenital goitre among 642 goitrous children; but this probably refers to districts which are definitely goitrous, for such a maximum has not been observed in other districts (Flesch and Winternitz). The comparative histological examination of the thyroid glands taken from healthy children in Berne and in the north German low-lying districts is of very great interest (Senschnied). This shows that the thyroids of Berne are not only larger and heavier than those of Germany, but that there are important differences in the details of their structure. The Berne thyroids have much smaller follicles, and frequently possess very large nuclei, rich in chromatin such as is encountered in goitrous nodules. The arteries also show peculiar changes. These characteristics are probably the result of the action of the goitrous poison which is still a hypothetical substance, some attributing it to drinking water, others to some infective material. They constitute the histological expression of the tendency towards the development of goitre.

We do not know whether the actual cause of congenital goitre is a transmission of toxic substances to the foetus, or whether it depends upon an inherited defect in the gland. Fürst suggests that congenital goitre is due to hereditary syphilis, but this has not

been proved. At any rate, enlargement of the thyroid gland is no ordinary symptom of congenital syphilis. English authors (Simpson, Hewetson, Macdonald, Forthergill) have reported cases of hyperplastic, parenchymatous, congenital goitre wherein the mother had been taking potassium chlorate during pregnancy. As the parents were free from goitre this affection in the child was attributed to the administration of potassium chlorate to the mother.

The anatomical changes which underlie congenital goitre are of various kinds. The following forms are recognized:

(1) Vascular goitre, or congenital telangiectatic goitre. This is mentioned first, not because it is most frequent, but because its histological structure shows that it is connected with the congestive swelling of the thyroid gland described above. It consists essentially in a dilatation of the subepithelial capillary layer. As is stated by Hesselberg considerable hyperæmia in the small glands of 2-4 gm. in weight may result from congestion, but in the larger



FIG. 31.—Congenital goitre.

glands up to 40 gm. in weight, hyperplasia of the whole vessel-system must be suspected in addition to passive hyperæmia. It cannot be stated precisely to what extent the parenchyma shares in this hyperplasia. Vascular goitre generally affects the whole organ which is of a soft homogeneous texture. Occasionally murmurs are audible over the vessels.

(2) Struma parenchymatosa, struma hyperplastica. The majority of congenital goitres belong to this form. There is an increase of gland lobules in the sense of normal growth. The alveoli are sometimes full of loose accumulation of cells, and sometimes are found with numerous round vesicles and long tubules filled with colloid, such as exists in the normal thyroid gland of the newly born (Hesselberg). In other cases congenital adenomas or epithelial neo-growths have been observed, which develop from embryonic, atypical vascularized gland formation

(Wölfler). The parenchymatous strumas may weigh 10 to 40 gm.; even greater weights have been known (100 gm.); but these cannot be true parenchymatous forms. The enlargement involves either the whole gland or isolated lobules; frequently one lateral lobe is particularly enlarged (see fig. 51).

(3) *Struma fibrosa* is the rarest form of congenital enlargement of the thyroid gland. Such a case was observed by Demme.

(4) *Struma cystica et colloides*. Cystic and colloid goitres are extremely rare in the newly born. These, however, have been described, particularly by earlier writers, but probably the majority of tumours in the neck described as cystic strumas should be regarded as teratomata. Wölfler's anatomically examined case proves, contrary to the assertions of Virchow and Demme, that colloid goitre does take place in the newly born.

The third and fourth forms of goitre have no relationship to endemic goitre.

(B) CONGENITAL TERATOMATA OF THE THYROID REGION.

Teratomata of the thyroid region never arise from the thyroid itself, therefore, strictly speaking, they are not thyroid tumours. Nevertheless, the relationship to the thyroid is generally very close; teratomata are located either in the immediate vicinity of the thyroid or in its substance; but the distinction between glandular substance and tumour can be easily shown. The development of the thyroid gland is generally interfered with by the large neoplasm. Isolated parts remain undeveloped; in fact, cases have been described where the thyroid gland was entirely absent.

Teratoid tumours are almost invariably very large. Monstrous tumours, the size of a child's head, have been observed. They are surrounded by a collective tissue capsule and situated below the superficial cervical muscles, and are either median or lateral in position. The large cervical vessels, the nerves and the sternocleidomastoid muscles are often considerably displaced. The surface of the tumour is generally irregular and rough; fluctuation from cysts can frequently be found in places. The histological examination shows a medley of tissues derived from all three germinal layers: connective tissue, adipose tissue, cerebral tissue, cartilage, bones, transversely striated and smooth muscle, skin and mucous membrane with their glands, mucous glands, retinal pigmented epithelium, &c. Hairs and intestinal loops in these tumours have also been reported (Morand, Joubert).

The teratomata either originate from branchial canal remains or they must be regarded as hagerminal teratomata (*foetus in foetu*) (Poult). Their development occurs during the early period of fetal life.

The majority of cases described are in premature children. Hereditary factors seem to play no part; they also have no connection with endemic goitre.

The tumours sometimes manifest a malignant character, a disposition towards continued growth, and metastatic deposits in the lymphatic glands (Pupovac).

Congenital teratomata in the region of the thyroid gland are rare. In the very detailed work of Hunziker dating back to the eighteenth century, twenty-one such cases are described. Probably many of the earlier observed cases of congenital cystic and colloid goitres belong to this category. The works of recent years are mostly based on careful histological researches (Swoboda, Schneider, Poulf, Flesch and Winterroitz, Hunziker).

The danger of congenital enlargements of the thyroid gland consists chiefly in its relation to the trachea. Large tumours, in particular teratomata, may cause complete compression of the trachea and thereby destroy the viability of the foetus. The children, if they are not already born dead, die shortly after birth from suffocation. Small strumas may result in perilous symptoms of compression. The softness of the infant's cartilage, the narrowness of the tracheal lumen, the above-mentioned condition of the thyroid gland, whose lateral parts sometimes spread round the trachea, are factors which facilitate the occurrence of symptoms of stenosis. They generally appear soon after birth, during the first few days, probably through much exudation into the tissues, and congestive hyperæmia, which is found even in persistent strumas. The symptoms of stenosis are either continuous or come in periodical attacks. The children manifest the well-known symptoms of tracheal stenosis, inspiratory and expiratory stridor, and usually marked retraction of the elastic thorax and cyanosis. If help is not forthwith at hand, death from suffocation may occur with these attacks. The situation of the head plays an important part. As a rule, the dyspnoic symptoms are most severe if the cervical portion of vertebral column is bent forward, the gland exercising strong pressure on the trachea. Accordingly, they frequently occur when the child is put down or during sleep, when the head is apt to sink forward or sideways. Under such circumstances symptoms of suffocation can also occur, even if the dangerous first few days have passed (sudden death on the seventh day in one of Denme's cases).

In smaller goitres the symptoms are mostly of an insignificant nature, and consist merely of slight stridor and mild attacks of difficulty in breathing, which are easy to deal with or pass off on their own. Symptoms of stenosis may also be entirely lacking.

If the infant has survived the first week of life, the subsequent prognosis depends essentially on the nature of the tumour. In the case of teratomata, rapid growth is to be apprehended, although they may have produced no trouble at first; but ordinary congenital goitres usually decrease in size in the early weeks. We must assume that pregnancy substances act in a similar manner on the infant as on the goitrous mother; the goitres increase in size during pregnancy and diminish after delivery. The shrinking may be

so considerable, even in the case of very large goitres, that on subsequent examination of the infant a doubt may arise as to the accuracy of the original diagnosis of persistent goitre. It is sometimes very difficult to differentiate true goitres from temporary swellings.

The principal object of the treatment of enlargements of the thyroid is to prevent symptoms of suffocation. As hyperæmia plays an important part in the causation of suffocation, the application of cold compresses or of an ice collar is of good service (Firkus, Planclou). The symptoms of obstruction which occasionally come on after the first warm bath can be rapidly relieved by these means (Escherich). The application of leeches, or of mustard leaves to the legs has also been recommended (Nicod d'Arbent).

Attention to the posture of the head is another important method of relief for respiratory embarrassment. The neck should be slightly bent backwards, which can be best effected by placing a small cushion under the nape of the neck (Freund, Læcher). Langer suggests that the head should be bandaged back by strips of plaster, reaching from the forehead, over the nape of the neck, to the back.

If, despite this, signs of obstruction nevertheless occur, air must be supplied in other ways. The tumour may be lifted off the compressed trachea by the hand, and time may be gained in this manner, until operative interference has been decided on. There are no reports in the literature in regard to intubation in this condition. This procedure would require a great mastery of the technique in an infant, but is quite feasible and it would be quite worth while to try, especially as the smallest intubation tube would reach deeply enough into the trachea. All authors unanimously agree that tracheotomy is not to be recommended. It is a most difficult procedure in a newborn goitrous infant, even for an experienced surgeon; all the cases operated on have succumbed to pneumonia in the next few days. On the other hand, good results have attended some other forms of operative treatment. It may be sufficient to cut through the isthmus (Malgaigne, Spitz), which can be done by means of an angiostyle and a double ligature. The hæmorrhage in the cases treated in this way has been trifling. French authors especially recommend exs-thyropexy (Polisson, Planclou and Richard, Fabre and Thévenot). The thyroid is exposed, and freed from the pressure of the surrounding skin and muscles, liberated as far as possible from the trachea so that it presses on it no longer, and is finally fixed by suture to the upper end of the incision. The goitre subsequently shrinks and undergoes atrophy. The removal of congenital goitre (partial thyroidectomy) has also been undertaken (Schümmelhursch, Lugenbuhl, Fischer, Spitz). The infants recovered in three cases; Lugenbuhl's case died of pneumonia after tracheotomy. The time for performing the operation depends upon the intensity of the asphyxial symptoms. Fischer recommends that it be done as soon as possible after birth (late hour). The operations have so far been done without an anæ-

thetic. In the case of teratodermia, the only correct treatment is removal, but it is quite safe to wait until the end of the first, second or third year, if there is no vital indication to operate beforehand.

In the absence of threatening symptoms, expectant treatment is all that is required, in the hope that spontaneous diminution of the goitre will occur. It is not necessary to start drug treatment for a few weeks; but some begin iodine treatment with the newborn, either in the form ofunction, enemata or injections. Rühemann recommends the administration of small doses of sodiumtyrin in the milk. The application of unguentum cinereum may also be tried. First thinks he has seen good results, in a case which was supposed to be syphilitic, by treating the mother with mercury.

On the basis of the theory that goitre is caused by the transmission of a poison from the mother to the foetus, the attempt has been made to inhibit the development of a goitre in the infant, by giving thyroïdin to the mother as a prophylactic (Mosse and Catia). Schmid reports the case of a family wherein two children died in the first days as a result of congenital goitre. The third child was born free from goitre, after the pregnant mother had undergone thyroid treatment (1 tablet of thyroïdin, once or twice daily until delivery).

CHAPTER IV.

CIRCULATORY SYSTEM.

(1) Abnormalities in the Position of the Heart.

The abnormalities in the position of the heart within the thorax, viz., mesocardia and dextrocardia (with or without situs inversus of the other organs) are not usually dangerous if there are no other mal-developments; but there are other abnormalities which do threaten life. These latter are comprised under the term *Ectopia cordis*. In this condition the heart is usually maldeveloped and is displaced either in the abdomen, or to the front of the chest or abdominal cavity; the pericardium may or may not be involved in the displacement. In these cases there is usually a fissure or larger defect in the sternum; in the more severe cases this is associated with a fissure in the abdomen. These deformed infants may live several hours or days; cases have been noted wherein life was prolonged for forty-eight hours (Greiffenberg, Maltenci).

(2) Congenital Hypertrophy and Dilatation.

Cardiac hypertrophy, which is often found at the autopsies of infants is not necessarily always of truly congenital origin. Nevertheless, in some cases the amount of hypertrophy is so great that the causes which produce secondary hypertrophy have not had sufficient time to act, considering the brevity of the life of the affected

infants (Ellron, Hedingen). The case recorded by Simmonds is perfectly definite. It concerned an infant who had died during birth, but its heart was considerably enlarged and weighed 44 gm., instead of 24 gm. The valves were healthy. No explanation has hitherto been forthcoming for this congenital hypertrophy. Presumably some unrecognized circulatory disturbances must take place during fetal life. There is little probability in the explanation of the change as a diffuse myomatous formation.

The diagnosis of pure congenital hypertrophy of the heart has not yet been made in newborn infants. The infants appear quite normal at first. The physical demonstration of cardiac enlargement is attended by difficulties, because the edges of the lungs may cover over the heart. The diagnosis of congenital defect of the heart can only be excluded with difficulty, even when the organ is shown to be large and hypertrophied *post-partum*.

Acute dilatation of the heart may occur in asphyxiated newborn infants as a result of disturbances in the pulmonary circulation. The right auricle is primarily affected by this dilatation. This dilatation can be demonstrated by percussion in certain cases. Owing to the excessive pressure in the auricle the foramen ovale remains patent so that venous blood streams into the left auricle and great cyanosis supervenes. Ferreira recommends as the treatment of severe asphyxia, in addition to artificial respiration, the use of stimulants in order to strengthen the heart muscle (injections of camphor, &c.).

[3] Congenital Abnormalities in the Heart and Great Vessels.

(A) THE PERSISTENCE OF FETAL PASSAGES.

The following is the course of the fetal circulation: The arterial blood in the placenta flows through the umbilical vein into the body of the infant and enters the inferior vena cava via the ductus venosus. Here it meets with the blood returning to the heart from the lower half of the body. This mixed blood which is comparatively rich in oxygen flows into the right auricle and the stream is directed through the foramen ovale into the left auricle and finally into the left ventricle. Thence it flows through the aorta into the arteries which are distributed to the head and the upper extremities. The venous blood from these regions flows through the superior vena cava into the right auricle and thence into the right ventricle, probably without any considerable mixing with the oxygenated blood which comes from the inferior vena cava. From the right ventricle, the blood flows into the pulmonary artery and then through the ductus arteriosus into the arch of the aorta. Here it mixes with the oxygenated blood from the ascending aorta and flows into the lower half of the body. The venous blood flows through the umbilical arteries arising from the hypogastric artery, through the umbilical cord back again into the placenta.

After the cessation of the placental circulation and the establishment of pulmonary respiration, the ductus venosus loses its blood supply, and takes no further part in the circulation. The venous blood of the right ventricle which fed the ductus arteriosus, now flows into the pulmonary artery and thence into the lungs. It there becomes oxygenated and flows into the left auricle, via the pulmonary veins. The high pressure in the left auricle closes the foramen ovale, and the blood from the left auricle flows into the left ventricle, which thus receives the arterial blood of the pulmonary veins. The blood leaves the heart by the aorta and supplies the systemic circulation, which does not receive any venous blood from the right heart owing to the closure of the ductus arteriosus.

The forces which determine the closure of the foramen ovale are quite clear. The pressure in the right auricle falls because the flow from the umbilical veins has ceased, and the pressure in the left ventricle increases owing to the increased flow therein from the lungs. Failure of the foramen ovale to close, owing to abnormal conditions of pressure, is also intelligible; but the factors which determine the closure of the ductus arteriosus have given rise to much discussion.

The most widely accepted explanation is that of Strassmann, who states that the ductus arteriosus opens into the aorta at an acute angle. At the moment of the establishment of pulmonary respiration, the pressure in the right heart and in the pulmonary artery falls, and the pressure rises in the left heart and in the aorta. The ductus arteriosus becomes less well filled and its opening into the aorta becomes compressed, and protruded forward in such a way as to produce a valvular closure.

Scharf assumes that the ductus arteriosus, with its slightly elastic, flexible walls, acts as the valve of a pipe owing to the suction effect of the aortic system—an assumption hotly contested by Strassmann and Roeder.

According to Thoma-Habenda the closure is caused as follows: In the fetal period the blood pressure is higher in the pulmonary artery than in the descending aorta, so that the current of blood flows from the pulmonary artery to the aorta; but soon after birth the direction of the current becomes reversed. Between these two periods there must be a moment when the pressure in the pulmonary artery and in the aorta is the same, so that the column of blood in the ductus arteriosus is at rest. At this moment, the muscular wall of the ductus arteriosus contracts up.

Kirstein takes up an intermediate standpoint. He acknowledges the importance of the equality of pressures in the aorta and pulmonary artery, and the consequent contraction of the wall of the ductus arteriosus, caused by its muscular and elastic elements, but he also attributes importance to the manner in which the ductus opens into the aorta. He also holds that some part is played by the change in posture of the heart, which follows the first respiration owing to the diaphragmatic movements, and by the protrusion which the pulmonary artery undergoes in its position.

The endarteritis obliterans, which finally determines the conversion of the ductus arteriosus into a fibrous band, can be recognized in its earliest stage, at the time of birth; but the obliteration of the duct does not occur until the third or fourth week (Roeder). The researches of Rauefuss, Strassmann, &c., have conclusively proved that the closure of the duct is not due to thrombosis. Thrombosis of the duct is pathological.

Summarizing the pathological essentials, we may say that the closure of the duct in newborn infants is mainly due to mechanical conditions, *viz.*, the changes in pressure consequent upon the alteration of the circulation from the foetal to the post-foetal state. The ductus is closed primarily owing to the displacement of its orifice and contraction of its walls. If it admits a probe during the first week, this cannot be considered abnormal; such permeability may persist into later life as an anomaly, without any clinical significance. The ductus arteriosus may fail to close at all (primary patency) or its closure may be arrested (secondary patency). The former is the more usual condition, and secondary dilatation of the patent channel may follow.

The causes which lead to patency of the duct are partly resistance in the pulmonary circulation (atelectasis), and partly abnormal conditions of pressure in the great vessels of the heart which may be due to their own abnormalities or to congenital anomalies of the heart. Defective development of the ductus is a supplementary cause, as must be assumed in the case of premature infants. Roeder states that the wall of the ductus, which is endowed with great contractility at the time of birth, loses its tension after the deviation of the blood stream. The time which elapses until complete obliteration of the ductus is critical for infants, for dilatation may occur owing to the flexibility of the obliterating channel, or aneurysm may develop, the intima may become lacerated, a dissecting aneurysm may form, or even rupture may occur.

The physical symptoms of a patent ductus arteriosus in later life are—a structure like a hood superimposed over the heart, which can be demonstrated by percussion and by a skiogram, and is due to the enlarged duct, a systolic murmur due to the passage of aortic blood into the pulmonary artery, accentuation of the pulmonary second sound, hypertrophy and dilatation of the right heart. In the newborn, however, these signs are but slightly developed. There need not be any severe cyanosis unless other cardiac abnormalities are present at the same time. At any rate, older children with definite evidence of a patent ductus arteriosus do not present any history of the symptoms just detailed. Escherich suggests that the pauses in breathing accompanied by attacks of cyanosis, especially seen in premature infants, may be due to an insufficient supply of blood to the lungs, in consequence of the circulation still proceeding through the ductus arteriosus. In such cases a murmur is not usually audible. Escherich, nevertheless, describes a definite systolic murmur which may occasionally be heard in the region of

the pulmonary artery during the respiratory pause, but which is not audible during the ordinary respiration and at the beginning of the attacks. The cause of this phenomenon is perhaps the rise of pressure in the pulmonary artery during the respiratory pause and a simultaneous drop in the arterial pressure; this increase in the difference between the pressures is followed by an increased current through the ductus arteriosus in the direction of the fetal circulation, and this provides the conditions which give rise to a murmur.

Newborn infants do not, as a rule, manifest any dilatation of the ductus arteriosus with aneurysmal changes. But definite distension of the walls of the ductus may occur, and result in rupture, as in the four cases described by Röder and Esser. There was a rent in the vessel, in one or more places, just as in a dissecting aneurysm. One of these infants died a few hours after birth from asphyxia in consequence of the cord being twisted round the neck. The second (breech presentation) died after two days with signs of intense dyspnoea; but the heart sounds were clear and distinct. The third, a well-developed child, with congenital goitre, died on the third day, rather suddenly, with symptoms of diffuse bronchitis. In the fourth case, the child was normal until the third day, and then began to breathe very faintly, without any obvious cause, and showed sclerodermatous changes in the skin. Death took place on the sixth day. There were symptoms of intense congestion in all these cases which may have led to changes in the conditions of the blood-pressure within the heart. Whereas Röder assumes that an increased pressure in the arch of the aorta forces open the valvular closure of the ductus arteriosus at its orifice into the aorta, Esser states that the increased pressure is in the pulmonary artery. Röder holds that death, in these cases, is not so much due to internal haemorrhage but to the mutual impact of the currents from the aorta and the pulmonary artery, and the consequent cessation of the circulation.

The foramen ovale is also closed, at first, through the blood pressure; it does not become sealed up immediately. If the pressure conditions become changed in diseases of the respiratory and circulatory apparatus, there is the possibility of an overflow of venous blood into the left auricle; this condition may explain the ease with which cyanosis may supervene in the newborn. As a rule the patency of the foramen ovale is only of importance if other cardiac abnormalities are present at the same time. It does not usually produce a murmur.

(6) CONGENITAL HEART DISEASES.

Congenital heart diseases may be due to some arrest in the process of development or they may be the result of a fetal endocarditis.

According to B. Fischer fetal endocarditis is always associated with diseases of the heart muscle, such as necrosis, infarction,

round cell infiltration, rheumatic indurations; the condition is therefore one of endomyocarditis. These diseases of the fetus may apparently be caused by acute infective diseases in the mother if they have appeared during the last two months of pregnancy. Thus Kockel describes mitral stenosis in a newborn infant whose mother had suffered from acute bronchitis during pregnancy. B. Fischer describes a case of congenital aortic and mitral stenosis due to an attack of influenza from which the mother suffered six weeks before her confinement. It is remarkable that the valves of the left side of the heart were affected in both these cases just as we see in acquired endocarditis of later years. Congenital changes at the orifices of the right side of the heart, as well as congenital abnormalities of the heart, have been attributed to infective diseases, especially syphilis in the parents. It is supposed that the inflammatory processes inhibit and disturb the normal development of the heart.

The majority of congenital heart diseases must, however, not be attributed to foetal inflammation but rather to malformations. This is evident from the purely clinical standpoint, for these diseases are frequently associated with other abnormalities in different parts of the body, such as degenerative changes in the nervous system and such constitutional abnormalities as mongolism for example. The actual cause is some primary arrest or disturbance of growth resulting in maldevelopment either in position or form of definite portions of the heart (Ziegler).

General Symptoms and Diagnosis.

The diagnosis of congenital disease of the heart in the newborn depends essentially upon two symptoms, viz., cyanosis and the presence of murmurs over the heart. The cyanosis is not due to congestion; it is caused by abnormal communications between the right and left side of the heart, resulting in the mixing of arterial with venous blood. The cyanotic aspect of the skin may be very intense; the skin and mucous membranes may present such a blue-black coloration as to justify the term "*morbus coeruleus*." The cyanosis may affect the entire surface of the body or only the peripheral part; it may be very intense for the first few days, but may begin to diminish considerably directly after birth.

Although cyanosis is an important symptom of congenital heart disease, it must not be forgotten that this symptom, in the early days of life, is very equivocal in its significance, and is very frequently caused by defective respiratory exchange in the lungs, resulting from disturbances in the respiratory centre or from pulmonary affections. The lung disease which is the cause of cyanosis, may, as mentioned above, be followed by the patency of the ductus arteriosus or foramen ovale, and this condition may determine an increase of the cyanosis. Finally, mention may be made of Winkel's cyanosis and similar conditions.

On the other hand, there are many congenital diseases of the heart in the newborn, which are not accompanied by any striking

cyanosis; so that the absence of cyanosis does not exclude a possible presence of cardiac abnormality.

As functional murmurs in the heart hardly ever occur in young infants, the presence of a definite murmur audible on auscultation must be accepted as a conclusive sign of a congenital cardiac abnormality, whether the murmur be loud (blowing, harsh or musical) or soft, as long as it can be confirmed with certainty when the infant is perfectly quiet. Some murmurs, e.g., the systolic murmur due to stenosis of the isthmus, can only be heard, or at any rate are heard better, on auscultation of the back.

Loud murmurs can be detected on auscultation of the fetal heart during pregnancy and therefore a diagnosis of cardiac disease may be made before the birth of the infant. Hoshino puts forward the following signs to differentiate this murmur from one which originates in the umbilical cord—the murmur is harsh and does not synchronize with the maternal pulse; it is constant in duration and intensity; as a rule both cardiac sounds are persistently absent, but occasionally only one sound is absent; the wide area of the abdomen over which the murmur is audible, but attaining a maximum intensity in the region of the fetal heart.

Important though a murmur be for the detection of cardiac disease, the absence of a murmur does not justify the conclusion that there is no heart disease, for the most extensive changes may occur in the heart and great vessels without the production of a murmur. This is quite intelligible, when one considers that a murmur only arises when the blood flows through a constricted part or through valves deformed by inflammation, or when eddies form anywhere in the blood-stream. But these conditions are not present in a congenital cardiac anomaly; the valves are not usually deformed and the abnormal channels of communication are wide. It happens sometimes that murmurs, which were hardly audible at first, manifest themselves plainly after a few days.

It follows, from the foregoing, that the diagnosis of "congenital heart disease" can be confidently made if a murmur is audible over the heart accompanied by a more or less definite cyanosis; even if the infant is not cyanosed the diagnosis is certain as long as the murmur is definite. A persistent and severe cyanosis in a lusty infant, who is not asphyxiated and who shows no signs of lung disease, suggests the presence of heart disease, with great probability, even if there be no murmur. If the cyanosis is intermittent, the diagnosis must remain in doubt. But if there is no cyanosis and no murmur, the diagnosis of heart disease cannot be made with certainty in newborn infants.

Dilatation and hypertrophy of individual chambers of the heart do sometimes occur on the first day, but, as a rule, do not appear until some time after the establishment of the post-fetal circulation, and they are, indeed, results of some obstruction to this circulation. Definite conclusions cannot, at this stage, be obtained either from percussion or from a skilgram. Percussion is only of diagnostic

value, if a murmur be heard, or if cyanosis exist—without any other cause for them. The same applies to accelerated respiration or pulse, to polycythæmia, to the low temperature which is so frequent in cyanotic infants, to certain cerebral symptoms (disturbance of consciousness, drowsiness, inadequate desire for drink, &c.) which are often noted in children with congenital defects and which possibly are connected with circulatory disturbances. A "heaving" pulsation of marked intensity in the region of the heart is not a frequent symptom in the newborn infant, but when it does occur it indicates with great probability the presence of a congenital abnormality of the heart.

In regard to the prognostic significance of the symptoms just described, it may be stated that the outlook is quite good in the case of murmurs which are not complicated by cyanosis; but one cannot foresee whether more serious symptoms will arise, if intercurrent disease should occur in the course of the infant's growth. According to Hochsinger the importance of cyanosis may be appraised as follows—infants born with a dark blue colour do not survive very long; if the cyanosis decreases after birth but does not disappear completely, the infants may live for a few months or even for years; if the cyanosis diminishes rapidly and disappears in the early days, the prospect of a prolonged life is quite good.

Special Pathology of certain forms of Cardiac Abnormalities.

The numerous possible combinations of congenital cardiac abnormalities render the diagnosis very difficult in any individual case, even in older children and in adults. This is the more true in the case of newborn infants, who rarely manifest uncomplicated typical forms of the characteristic symptom-complex, which comes gradually as a result of the demands upon the heart to accommodate itself to extra-uterine conditions. In describing briefly the most important types of congenital heart disease (based mainly on Hochsinger's statements) it is intended merely to indicate the direction which may lead to a diagnosis. As a rule, one must be content with recognizing that a congenital cardiac abnormality exists; if there are no physical signs it is impossible for the most experienced to make an exact diagnosis, and if some definite physical defect is diagnosed, the post-mortem often reveals a number of undetected abnormalities.

(1) DEFECTS IN THE WALL SEPARATING THE TWO SIDES OF THE HEART.

(a) Defects in the Septum.

A frequent abnormality. Sometimes occurs by itself, but more often combined with other abnormalities, especially pulmonary stenosis.

Symptoms. Harsh systolic murmur in the middle of the cardiac area (Roger) only if there is a difference in pressure between the two ventricles and the opening remains patent during the contraction of the chamber. The murmur is often absent. Second pulmonary sound usually accentuated. Severe cyanosis not usual.

The prognosis is generally good.

(b) Patent Foramen Ovale (see above).

(2) ATRESIA AND STENOSIS OF THE LARGE ARTERIES.

(a) Pulmonary Stenosis.

The stenosis may be at the ostium or in the conus. Pulmonary stenosis is one of the most frequent and most important of congenital cardiac abnormalities. It is often associated with septum defect, patent foramen ovale and persistent ductus arteriosus.

Symptoms. Systolic murmur over the pulmonary artery; but this may be absent or a late symptom. Weakening of the second pulmonary sound, unless the ductus arteriosus remains patent at the same time. Considerable cyanosis as a rule. The infants are often apparently stillborn, but they revive quickly. The duration of life averages ten to twelve years, but may be longer. General condition often very good, despite the intense cyanosis.

Second sound remarkably weak in pure stenosis, or when associated with a patent foramen ovale. The murmur is not conducted into the vessels of the neck.

If associated with septum defect, the pulmonary second sound is clearer, but not accentuated. The murmur may or may not be conducted into the vessels of the neck.

If the ductus arteriosus remains open at the same time, the second sound is greatly accentuated and intensified, and the murmur is distinctly conducted into the carotid and subclavian arteries.

Atresia of the pulmonary artery is rare. Bach reports a case which was associated with stenosis and insufficiency of the tricuspid valve and persistence of the foramen ovale and ductus arteriosus. There was cyanosis and dyspnoea, but no murmur. Death occurred in seven weeks.

(b) Aortic Stenosis.

The stenosis may occur in the ostium, the conus or the isthmus. The systolic murmur which is heard as in the corresponding stenosis of acquired heart disease, may be absent in severe stenosis, if the ductus arteriosus or the septum remain open at the same time. In cases of isthmus stenosis, the murmur is often heard best in the back.

In atresia of the aorta (complete obliteration of the first portion) there is usually a vicarious dilatation of the pulmonary artery, a

widely patent septum ventriculorum and an open ductus arteriosus. The duration of life is rarely more than a few weeks.

(3) CHANGES AT THE VENOUS ORIFICES.

Congenital atresia, stenosis and insufficiency may occur both at the mitral and tricuspid valves. Stenosis and insufficiency present the corresponding symptoms of acquired heart disease, but they are almost always associated with other abnormalities (especially defects in the septum) which affect the clinical picture.

According to Külm, congenital atresia of the tricuspid orifice causes intense cyanosis, attacks of suffocation, oedema, &c., starting at birth; there is also a loud systolic murmur, and enlargement of the heart towards the left just as in pulmonary stenosis. The prognosis is bad; but there is a possibility of life lasting several years, the oldest recorded case having reached twenty-seven years. Spolverini and Barbieri describe a case of mitral atresia with a systolic murmur, with death at the fortieth day. In atresias survival is naturally only possible when abnormal communications are present between the two sides of the heart.

(4) ABNORMALITIES OF THE LARGE VESSELS.

(a) *Persistence of the Ductus Arteriosus.*

Symptoms.—Dullness (see above). Heart sounds clear, or systolic murmur with maximum intensity at manubrium sterni, more rarely a diastolic murmur, conducted towards the upper thoracic opening, into the axillae and to the back. Accentuation of the second pulmonary sound. There is not usually any cyanosis in pure cases. This abnormality is in itself compatible with a long duration of life.

(b) *Transposition of the Roots of the Large Arteries.*

Aorta in the place of the pulmonary artery and vice versa. The vessels may arise from one chamber, or the aorta may arise from both chambers, riding over the septum. Often combined with defects in the septum, patent ductus arteriosus, stenosis of the cardiac orifices. Symptoms very indefinite. Usually much cyanosis; if the transposition is complete there is accentuation of the second sound at the base, in consequence of excessive pressure in the pulmonary artery. Systolic murmur below centre of heart in consequence of septum defect. Duration of life limited; death usually within the first six months.

(4) Disturbances in the Cardiac Mechanism.

The observations hitherto made on the behaviour of the cardiac mechanism in newborn infants are too few for definite conclusions concerning the frequency with which derangements occur. Several

authors have described a deep Jp notch in the electro-cardiogram of adults with congenital heart disease, but this is of no special value in regard to newborn infants, because, at this age, the Jp notch is very deep in normal conditions (v. p. 46). Hecht found in the case of a newborn infant with heart disease that Jp = 100 per cent. of J, a value which comes well within the range of normal.

It is not known whether disturbances of impulse conduction in the heart of the newborn occur in various general diseases as a result of septic or toxic influences. The following case of congenital disturbance of conduction, the etiology of which is obscure, and which ended in recovery, is therefore of great interest.

The infant weighed 3,500 gm. and was a well-developed child of a healthy primipara; there was definitely erythema on the first day, which had attracted the attention of the accoucheur who auscultated over the uterus; the fetal heart beating in a triple rhythm, otherwise physical condition at heart normal; no abnormal conditions shown by Röntgen rays. No cyanosis, no dyspnea. The arrhythmia, sometimes combined with pronounced bradycardia (70), was distinctly observable up to the sixth day.

On the second day Dr. A. F. Hecht made an electro-cardiographic examination, using the usual three leads. At the same time respiration was registered. The frequency of pulse fluctuated between 115 and 120, the frequency of respiration amounted to 60. The interval of the ventricular systoles increased sometimes to double, to 200–240°, whereas the normal interval is 180–210°. This variation of the ventricular systole followed sometimes after 100 beats, so that a kind of bigeminy arose, then it was absent again through 30 beats and longer. A continuation of the abnormally long pulse period with the phase of expiration, such as is found with arrhythmia, did not occur; probably a blocked auricular action (Vorhofsknoten) preceded every miss of the ventricular systole. It followed the auricular systole at a very short interval (102°). Whether it was a premature extra systole or, less probably, a reverted action from the ventricle to the auricle, could not be decided. In any case it always happened when the ventricular systole had not finished; therefore its fluctuation was superimposed on the back flow (Fig. 72). It could not be decided definitely whether heart muscle conductivity was absent because the conductive capacity could not be so quickly recovered, corresponding to the persistency of the extra systole, or whether there was actually at the same time a disease of the bundle of His.

On the seventh day of life no further arrhythmia could be found. In the third week the child was again electro-cardiographically examined; normal conditions again existed. Arrhythmia had disappeared. Action of the heart was active (120–160). After difficulties at first in taking the maternal breast the child developed normally and increased in weight from 3,100 gm. (15th day) to 3,300 gm. (27th day).

(5) Acute Endocarditis and Pericarditis.

Acute endocarditis, rheumatic as well as septic, practically never occurs in the newly born (Lempp). A case of septic endocarditis, described by Czerny, appears to be the youngest (one month old).

Among diseases of the pericardium only purulent pericarditis has hitherto been observed in the newly born; it also is rare. It is usually a metastatic suppuration in the course of a pyæmic disease. Sometimes it is a complication of purulent pleurisy. The author observed purulent pericarditis as the only metastasis in a child of eight days, with purulent meningitis (bact. coli). The disease runs its course without any characteristic symptoms.

Those formations of nodules on the cardiac valves of newly born

known as "hematoma of valve" are not hemorrhages, but are derived from dilatation and cysts; they are residues of the process of evolution of the embryonic vascular tissue at the cardiac valve (Berti, Fahr). Probably no clinical importance is attached to them.

(6) Diseases in the Region of the Blood-vessels.

Only one form is known of diseases of the walls of blood-vessels, *viz.*, the syphilitic (*vide infra*).

Thromboses occur during the course of septic conditions, and in marasmus; newly born Hochsinger observed an inflammatory thrombosis of the art. thoracica longa in a newborn child with pneumonia that led to cutaneous necrosis in the region of the artery, and Rauchfuss observed a suppurating thrombosis in the ductus Botalli. Wirz describes a thrombosis of the inferior vena cava and of a renal vein in a child of fifteen days, causing hæmaturia and hæmorrhages in the abdominal cavity; ætiology unknown. See also p. 335.

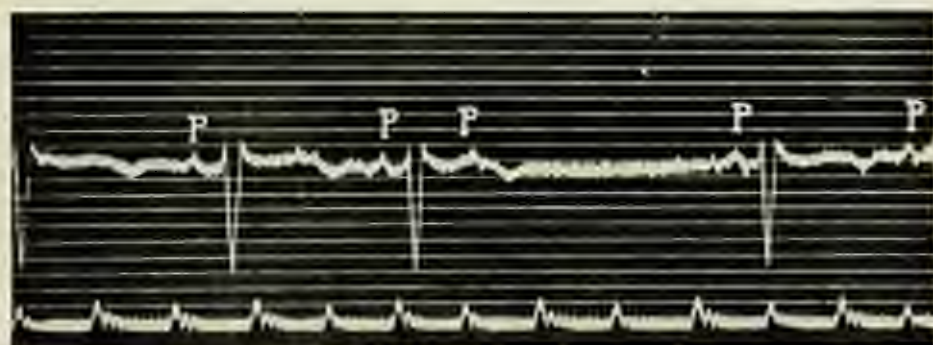


FIG. 57.—I, aVL, & M.V., = 2 cm. There is an auricular depression (post-systolic δ) in the after wave of the third beat, which is not followed by any ventricular wave (blocking). The intervals between the auricular systoles consist of 0.55, 0.51, 0.49, 0.50, 0.52 $\frac{1}{2}$; the pause is thus not completely compensated for. (The time marking corresponds to $\frac{1}{5}$ and $\frac{1}{10}$.)

CHAPTER V.

BLOOD.

Though material for observation is so abundant in the normal hæmatology of the newly born, yet our knowledge as to the pathological changes in the blood during this period is very scanty.

The majority of diseases of the blood in the newly born is certainly acquired, even if the disposition may be in many cases congenital. In some forms of anaemia in the infantile period, originating from a congenital disposition, from congenital "weakness of the blood-forming organs," or "functional inferiority of

the bone marrow" (Benjamin), one finds in the previous history that the children have often been pale and sickly from birth. General pronounced anaemia is uncommon during the first weeks of life. They are generally forms of secondary anaemia after haemorrhagic diseases (melaena, suprarenal capsular haemorrhages, &c.) in the course of septic conditions, in syphilitic children, &c. Such forms of anaemia are sometimes extremely severe.

Concerning the occurrence of congenital leukaemia, cases of hydrops foetus universalis have been described in earlier writings, which have been attributed to congenital leukaemia on the basis of the post-mortem results of children already born dead or dying immediately after birth. In the case of Jaksch, Eppenger found changes which correspond to a "leukæmoid, if not a completely leukæmic condition"; in the case of Sanger the proportion of white corpuscles to red was 1 : 3; in the case of Siefert nodular masses of leucocytes were found in all the organs in connection with the vessels, also free in the connective tissue. The mothers were all free from leukaemia. On the other hand, women suffering from this disease did not bear children affected with it, so that there is no evidence for the transmission of leukaemia *in utero* (Sanger).

Recent researches show the improbability of the existence of the connection, which was formerly assumed to exist, between hydrops foetus universalis and leukaemia. Schridde and Fischer found numerous myeloblasts in the blood of fetuses with hydrops. In a case of Rautmann's there was great reduction of all the leucocytes; in his opinion there was a pathological proliferation of the erythroblastic tissue, probably as a result of the metabolic disturbance caused by maternal nephritis (*vide infra*).

Zuccola reports a case of haemorrhagic diathesis which developed in the course of a rapidly progressive disease which occurred in the early days of life, with vomiting and diarrhoea. It was attended by intermittent pyrexia, and death occurred in the fourth week. Blood examination showed that the red corpuscles were diminished to 1,000,000 and the white corpuscles were increased to 38,000. A differential blood-count corresponded to that of lymphatic leukaemia.

Lommel reports a case whose blood count on the twenty-eighth day indicated a lymphatic leukaemia, viz., 1,290,000 white corpuscles, mainly lymphocytes, and 1,282,000 red corpuscles. The child was very pale and thin and had a very large splenic tumour; the latter was apparently present at birth, because both the mother and the midwife were struck by the distension and hardness of the abdomen, which continued to increase during the first weeks. The explanation of this case remained obscure; the parents were healthy, and there had been no infective disease during the pregnancy.

Pollman describes a case which was very probably a congenital disease of the blood; he termed it "spleno-medullary leukaemia." The child had purpuric spots at birth. When 14 days old a considerable enlargement of the liver and of the spleen was detected. The red corpuscles numbered 2,500,000; the white corpuscles were

greatly increased, the proportion of white to red being 1 to 8, being mostly mononuclears with rather large nuclei. The child soon died and the post-mortem showed that there was congenital heart disease (endocarditis of the tricuspid area). The author assumes that there was some infection during foetal life. Neukirch reports an example of a great increase of myelocytes in a definitely septic case. These conditions of the blood are intelligible in view of the newer conception of the essence of acute myelogenous leukaemia as a "general infection wherein the blood-forming organs participate largely" (Sternberg).

SPLEEN.

Absence of the spleen may occur as the only abnormality, or it may be one of many. In the former case it has no clinical significance; persons suffering from this abnormality may reach a ripe old age (Krauss).

Any septic disease may produce an acute swelling of the spleen; but chronic tumours are especially prevalent in congenital syphilis, diseases of the blood, hydrops foetus universalis and congenital malaria.

We do not know how far the recently established functional importance of the spleen is concerned in the metabolism of newborn infants, nor do we know whether disturbances in the splenic function occur within the early days of life.

CHAPTER VI.

GENITO-URINARY TRACT.

Diseases of the Urinary Organs.

The kidneys, like the liver, may be affected, during the course of various general diseases, by diverse toxic and infective factors. The damage presents itself at the autopsy in the form of parenchymatous or fatty degeneration of the cells, just as in the case of the liver.

The clinical symptoms of kidney diseases of the newborn (albuminuria, presence of casts), like those of older infants which are caused by alimentary intoxication, are not always explained by the changes which the pathologist usually identifies with nephritis. Even if the condition of the urine corresponds to "parenchymatous nephritis," the anatomical diagnosis does not, as a rule, proceed beyond renal degeneration. The proposal of Friedrich Müller to designate all inflammatory and degenerative changes in the kidneys by the term "nephrosis" is particularly appropriate to the clinical requirements of early infancy.

It is at present quite impossible to offer an accurate classification of kidney diseases which are not at all rare in the newborn. The

clinical and pathological records are too few and too sufficiently precise. The modern methods of testing the renal functions have hardly been applied to the newborn, so that accurate clinical observations are wanting. The ordinary symptoms of renal disease, such as albuminuria, hæmaturia, casts and oedema are not very helpful in the newborn.

Most newborn infants have slight albuminuria, so that the presence of this symptom is equivocal. Neither must one assume that "nephrosis" exists, because there may be a few casts and red corpuscles in the sediment during the first few days. One must not conclude that there is any morbid condition unless these symptoms are severe and persist unchanged beyond the first three days. It is very probable that the physiological albuminuria of the newborn, which is usually slight and transitory, may be aggravated and prolonged by various infectious, chemical and physical causes; the transition from the physiological to the pathological is very gradual.

It is very exceptional to encounter well-developed classical signs of nephritis in the newborn; albuminuria of more than $\frac{1}{2}$ per mille is very rare. One of the most important forms of nephritis in the newborn, the syphilitic, will be discussed later on. From the theoretical standpoint, great interest attaches to the question of the influence of the nephritis of pregnancy and the eclampsia so often associated with it, on the kidneys of the child. There are two possibilities in regard to the transference of substances which may damage the kidneys of the fœtus. The toxic material which caused the maternal nephritis may be transferred to the fœtus, or the toxins retained in the maternal blood owing to the nephritis may gain access to the fœtus. If, owing to the mother's illness, the infant becomes diseased in a manner which is clinically recognizable—and this is by no means always the case—the results are usually of a degenerative character. The cell degeneration may be very intense, but it is very rarely that any definite nephritis occurs in the infant, coincidently with kidney disease in the mother. The author has repeatedly found that the urine of the infant of a nephritic mother is quite normal, even if the mother nurses the infant during the period of the full intensity of her nephritis or during its subsidence. The condition of the urine may be normal, even if the infants themselves suffer from convulsive attacks (Esch). Nevertheless a number of positive observations are to be found in the literature. Weisswange and Rietschel report a case, wherein the infant of an eclamptic mother had been in a somnolent condition since birth but had no convulsions. On the fourteenth day the existence of a hæmorrhagic nephritis with 1 per mille albumin was noted. The renal symptoms subsided after ten days. Gruler also observed nephritis in the child of an eclamptic mother, who died from convulsions when five days old. Goodall noted an albuminuria of eighteen days' duration in the infant of an eclamptic mother, and he assigns great importance to maternal eclampsia as a cause for infantile nephritis. Sizenfrey records the case of a mother with

acute nephritis and oedema; she was delivered of twins who died soon after birth, but they both had acute nephritis with oedema and ascites. Hydrops foetus universalis also seems to have some connection with the nephritis of pregnancy (*vide infra*). Finally, there appears to be some association between hydramnios and maternal nephritis and changes in the infantile kidneys (Læven).

Chronic nephritis in the mother may apparently exert some influence on the child; at any rate, this would appear to follow from the observation of Hellendal, who reported a case of contracted kidneys in an infant, aged 6 months, whose mother suffered from nephritis. Democh and Fröhlich also describe cases of primary contracted kidneys—probably congenital but certainly not syphilitic—in young infants. One of the cases of the latter author is of special interest for the pathology of the newborn, because the diagnosis was established in the first month.

A comparatively large number of cases of "nephrosis" in newborn infants is of infective origin. Mensl reports seventeen cases of nephritis in infants ranging from 10 to 14 days old, in all of which he assumes that there was secondary infection after diseases of the respiratory or digestive tract.

The occurrence of haematuria and haemoglobinuria is discussed elsewhere. Here only reference will be made to the so-called congenital haematuria (hereditary and familial). The disease consists of sudden appearance of blood in the urine, a condition which has occasionally been observed in the first week of life (Aitken). Finkelstein suggests that it has some connection with paroxysmal haemoglobinuria.

Finkelstein reports a case of thrombosis of the renal veins. The infant, who was suffering from separation of the umbilical cord, showed symptoms of a severe toxæmia with diarrhoea, in the second week. On the fourth day a definite enlargement and distinct painfulness of the left kidney were observed. The urine contained a little blood and was highly albuminous. The post-mortem showed a complete infarction of the left kidney, and the vein contained recent loose thrombi from which streptococci could be isolated.

Cases of stone in the kidney have not been observed in the newborn period (Bokay). Some authors, however, have associated its occurrence with infarction of the kidney in the newborn, and they have recommended as a prophylactic measure the administration of enough fluid in the first days of life to assist in the excretion of the infant (Zuckerkindl). The unequal geographical distribution of infantile lithiasis shows that there is no cogency in the view that its prime cause is the "exsiccation" of the newborn infant.

Congenital *hydronephrosis* sometimes occurs because the orifice of the ureter opens into the pelvis of the kidney at an acute angle, so that when the pelvis is very full the orifice of the ureter closes like a valve (Eröss). This form of valvular hydronephrosis may be intermittent in character. In other cases the condition is caused by some congenital obstruction in the urinary passages, or by abnormalities in position or malformation of the kidneys (Roesshke

kidney). The hydronephrosis may be so great at birth that the distension of the abdomen may cause difficulty in labour, but as a rule the enlargement of the abdomen is gradual (Sherlaw). The disease may be uni- or bilateral. If the diagnosis is doubtful, it can be settled by an exploratory puncture.

Congenital hydronephrosis possesses certain clinical resemblances to congenital cystic kidneys. The latter contain numerous cavities of varying size and usually circular in shape, which are retention cysts consequent upon the blocking of the urinary tubules by masses, or by inflammatory products. These cysts may also be the result of some defect in the development of the tubules. These cystic kidneys are often recognizable at birth as distinct tumours. They are usually bilateral and are associated with other mal-developments.

Bolt reports such a case, diagnosed at birth as a cystic kidney. At first an extra-abdominal left-sided tumour was detected, which seemed to resemble the spleen, but a right-sided tumour was also detected very soon. The urine contained no albumen. Death occurred at six weeks. The pathological process was one of neonatal nephrosis with cyst formation, just due to retention and not due to contraction of connective tissue.

As tumours of the kidney occur with comparative frequency in children during the first year, we may assume that they are often of congenital origin, although they may cause no symptoms in very early infancy. It is well known that small innocent tumours give no clinical signs; in the newborn congenital fibromata of the kidney are not usually larger than a lentil seed (Rucker). Malignant tumours of the kidney in the foetus and newborn belong to the embryonic type of mixed tumours. They are usually described in the literature as adeno-sarcomata. Weigert reports a case of bilateral adeno-sarcoma of the kidneys in a still-born child. These tumours grow rapidly and are fatal in the course of a few weeks or months, although they show no special tendency to the formation of metastases. If one of these unilateral tumours be removed soon enough, the prognosis should not be absolutely hopeless (Steffen). The clinical diagnosis of congenital tumours of the kidney is, at first, always obscure, especially if they are cystic. The differentiation of cystic mixed tumours from cystic kidneys is hardly possible at first. The general diagnosis of "renal tumour" is rendered difficult by the fact that congenital tumours of this region often affect the adrenals and not the kidneys.

Adrenal tumours may be unilateral or bilateral, and may be sarcomata, adeno-sarcomata or hypernephromata (Makai, Marthen and Shukowski). It has been noted that adrenal tumours are frequently associated with tumours of the liver. Cystic tumours may also occur in the adrenals.

It is not necessary to go into further detail concerning the different abnormalities of the kidney (absence, hypoplasia of one with hypertrophy of the other, pelvic kidney and other deformities). A mere reference to the so-called *congenital floating kidney* will suffice. Finkelschein holds that if a kidney in a newborn infant moves

on respiration, i.e., its lower pole can be felt on deep breathing, it must be regarded as in a condition of ptosis. He states that it is almost always possible to palpate the lower half of the kidney with some practice. This physical sign is not unimportant.

The pelvis of the kidney, the ureter and bladder are not frequent sites of disease in the newborn; at any rate, our knowledge of them is very defective. The occurrence of infective disease in the excretory apparatus, e.g., pyelocystitis, is naturally quite as likely in newborn infants as in older children; indeed it would appear to be a more probable contingency in view of the comparatively great frequency of general infections in the early days of life, but clinically there is little evidence thereof. Kowalewsky and Moro have described the case of the youngest patient with pyelitis and cystitis. The infant was below normal weight and feeble, and passed urine containing much blood and albumin on the eighth day. The deposit contained numerous leucocytes and *Bacillus coli*. Death occurred on the eleventh day of life (coli infection).

We have already referred to the development of valves in the region of the ureter which may give rise to hydronephrosis; but mention must also be made of various abnormalities in the lower orifice of the ureter, e.g., uretero-vaginal, uretero-uterine and uretero-urethral atresia.

If the allantoic duct, which passes through the umbilical ring, remains open, an umbilical fistula results and urine escapes therefrom (fistula of urachus); this condition is caused by some disturbance in the development of the allantois into the bladder and urachus, or by intra-uterine retention of urine due to obstruction in the region of the urethra. Spitz recommends the subcutaneous injection of methylene blue in order to confirm the diagnosis; if there be any communication with the urinary bladder the fluid which escapes from the fistula will be coloured blue. As a fistula of the urachus involves the risk of infection of the urinary passages, it should be removed as soon as possible, but it is not desirable to undertake a plastic operation in the early weeks of life. If the lumen of the fistula is narrow, cauterization with nitrate of silver often suffices to close it. If the lumen is only partially permeable "urachus cysts" lined with cylindrical epithelium are apt to develop—mostly in the lower third (Wutz).

The malformation in this region which is of the greatest practical importance is *ectopia testis*. Like other malformations resulting in clefts, it must be attributed to some arrest of development. The mucous membrane of the posterior wall of the bladder with the orifices of the ureters protrudes as a reddish tumour above the symphysis, through the median abdominal cleft, which may reach as high as the umbilicus. The cleft usually also involves the urethra and the external genitals; in boys the penis is rudimentary and epispadias, the scrotum is divided and the testicles undescended. In girls the labia and the clitoris are cleft and the commissure is absent. Treatment, by means of operation, is never completely satisfactory.

and operative measures should be postponed until infancy is well advanced (Spitzzy). The conditions can be made more favourable for a subsequent operation by wearing a compression apparatus. The most important point is to prevent an ascending infection by means of scrupulous cleanliness.

Diseases of the External Genitals.

(5) MALE GENITALS.

Hydrocele is generally due to some congenital cause, although the condition may not be manifest in the period of early infancy. The processus vaginalis often remains patent, and the fluid secreted



FIG. 53.—*Hydrocele and hematoma in the region of the right spermatic cord of a child 7 days old.*

by the serous surfaces issuing from the peritoneal cavity frequently flows into it. But typical hydrocele is very often seen in infants at birth. Treatment is unnecessary at first.

Fig. 53 represents an extremely rare case of *hematocele* of non-traumatic origin.

The abnormalities in the position of the testicle are not sufficiently obvious to be recognized in the newborn. Spitzzy points out

that in many newborn infants the inguinal canal, for a long time, permits of the return of the testicle into it. The influence of cold or the provocation of the cremasteric reflex causes the testicle to be drawn up into the inguinal canal, even as far as the abdominal cavity. When these causes cease to act the testicle reappears. The scrotum is remarkably small in these infants, and Spitzzy considers this to be a result and not a cause of the malady. In most cases the testicle acquires its proper position owing to its increasing weight in the course of its growth. Such terms as "retention of testis" (abdominal or inguinal cryptorchism) and "ectopia testis" (scrotal or perineal) can only be used justifiably of the newborn if the organ remains permanently in the abnormal position. Even in these rare cases operative interference should not be undertaken in the first few weeks, although it is particularly necessary in cases of inguinal testicle.

An abnormal position of the urethral orifice or clefts of the urethra are termed *epispadias* if they occur on the upper surface of the penis (vesical epispadias, penile epispadias, epispadias of the glans), and are termed *hypospadias* (H. scrotal, penile, glandular) if they occur on the under surface of the penis. The operative treatment of these deformities should not be undertaken until the child is a few years old.

Atresia of the urethra, which is a very rare condition, is of more practical significance for the pathology of the newborn. Severe deformities (e.g., complete absence of the urethra when the penis is present or defective) are of less significance. They are usually associated with abnormal openings of the urethra. An imperforate glans penis is of more importance; it usually depends upon some obstruction of the external opening of the urethra by means of membranous adhesions. In these cases the glans presents no indications of a urethra beyond a shallow blind depression (Langstein). If it is not a case of a harmless superficial agglutination of the external orifice, that may be broken down by means of a probe, operative treatment must be undertaken immediately after birth, in order to prevent retention of urine. Congenital urethral strictures have also been described; they may give rise to haematuria (Churchman).

For the newly born male *phimosis* is, up to a certain degree, a physiological condition. Between the interior preputial membrane and the glans penis epithelial adhesions are always present, which gradually loosen in the course of the first few months or years. But a congenitally too long and too narrow prepuce or the narrowness of the external preputial opening may be very unpleasant, even to the newly born, and create disturbances in the evacuation of urine; this is, however, not frequently the case. Operative or other mechanical methods are generally postponed to a later period; but one should attempt to remove the phimosis during the first year, for it is nearly always possible without an operation by merely forcibly pushing back the foreskin.

(b) FEMALE GENITALS.

The appearance of germs in the genital secretion may justifiably be regarded as a physiological process, the same as the invasion of the oral cavity or the intestinal canal with bacteria. Aetiological importance is not attached in the first days to this invasion of bacteria for the existence of disease.

As equally physiological must be regarded the swelling and mucous secretion of the vulva, which is invariably found, in a more or less marked degree, in all newborn girls during the first week. If the external genitals are inspected, a considerable succulence of the labia majora is noticed. If the latter are unfolded the labia minora and the clitoris are often observed to be relatively enlarged, and more or less abundant white, viscous, mucous secretion (somewhat less consistent in the following days) is observed at the mucous membrane of the vulva, sometimes coming out of the vagina through the hymen.

Epstein regards this as a manifestation of a widespread process of desquamation of all the integuments and their invaginations, which takes place already during foetal existence, and continues after birth owing to the changed state of existence and the external irritants affecting the newly born (Langstein). It is possible that in the genesis of this vulvo-vaginitis desquamativa neonatorum and genital oedema, pregnancy substances also, in Halban's sense, play their part. It must be emphasized, from a practical standpoint, that the above-mentioned processes must not be considered as pathological, and accordingly must not be treated as such. Frequent drying and removal of the secretion is to be recommended as a preventive of secondary infections. Pronounced vulvitis very rarely develops from these conditions (Noack).

Neisser, who examined bacteriologically the genital secretions of newly born girls, obtained the following results: The secretions of the vulva and vagina of newly born, that were not bathed, remain sterile if the secretion after birth is removed with aseptic precautions, the former up to 2 or 3 hours, the latter up to 12 hours. From the 7th to the 8th hour the vulvar secretion always contains germs, intestinal bacteria, chiefly of the coli group, but also staphylococci and streptococci. The germs appearing in the vagina after the 12th hour generally tend to disappear under the influence of the automatic purification of the vagina. In the flora of the vagina Döderlein's vaginal bacillus predominates. The first bath has an expediting influence on the appearance of germs in the genitals, but the occurrence of the vaginal bacillus is in no way influenced. Infection of the liquor amnii with premature rupture of the fetal membranes may expedite the appearance of germs.

Gonorrhoeal infection is by far the most important form of vulvo-vaginitis even in the newly born. If, in comparison with gonorrhoeal conjunctivitis, it rarely occurs, the reason may be that the opportunity for infection is less favourable. As Aichel particularly points out, the genital passages of the child pass through in a few minutes, whereas the head often remains hours in these parts which are so liable to infection. In the majority of cases gonorrhoeal infection of the vulva would be less likely to happen during birth

than after it. In the cases observed by Koblandk and Aichel the disease appeared at the end of the first week and in the second, and manifested itself in marked swelling and redness of the external genitals, and discharge of yellowish pus. Both authorities also observed hæmorrhages, a special disposition of the newly born to such being attributed as the reason; in older infants, vaginal hæmorrhage or the extension of the process to the uterus is not a part of the clinical picture of gonorrhæal vulvo-vaginitis. Extension to the urethra and bladder is a much more likely complication. But even without this the prognosis is not very favourable, because the disease lasts a long time, tends to recur, and thorough treatment is very difficult. Nevertheless, complete cure may take place within a few weeks. Epstein recommends the instillation of a 2 per cent. solution of nitrate of silver into the vulva as a prophylactic measure. If the mother is not suffering from gonorrhæa, modern methods of cleanliness should suffice to prevent the occurrence of the disease. But even if the disease exists, cleanliness is the first desideratum, viz., thorough washing of the genitals and removal of the discharge by means of a weak solution of permanganate of potash, applied with a cotton-wool swab. The mucous membrane of the vulva may be painted with a 2 per cent. solution of silver nitrate or a solution of protargol or sopsol may be instilled. It is better not to employ vaginal irrigation, in view of the fact that the disease has little tendency to spread in that direction. As regards vaginal hæmorrhage (*vide infra*).

A few cases of prolapse of the uterus in newborn infants have been recorded. This very rare affection has apparently occurred in children with spina bifida (Krause, Hanssen), although Radwansky has seen a case in a child who was otherwise quite healthy. The prolapse is seen immediately after birth as a round protruding tumour between the labia, or may not be evident until a few days have passed.

Congenital abnormalities in the region of the external opening of the urethra are comparatively rare. They include clefts in the urethra and clitoris described as epispadias and hypospadias, abnormal opening of the urethra into the bowel or into the vagina.

Atresia in the region of the external genitals is of some practical importance. Closure of the vagina by an imperforate hymen is called atresia vaginae hymenalis (Breisky, Bunzel, Commandeur). In these cases a tumour of the size of a cherry or pigeon's egg, with a grey waxy surface, protrudes between the labia when the child cries. Pressure on the tumour reduces its size. If it is incised, or if it bursts spontaneously after a few days, a profuse amount of milky-mucoid vaginal secretion escapes.

Epithelial adhesions of the labia majora and minora are termed atresia vulvæ. In certain cases this condition leads to retention of urine and of mucus. It is easily removed by breaking down the adhesions with a blunt probe.

A congenital cyst of the hymen may also cause disturbances of micturition (O. Müller).

It should be stated here that certain forms of retention of urine may occur in the first few days of life both in male and in female infants, although no mechanical obstruction exists. Possibly they depend upon the failure of the distended bladder to elicit a reflex for emptying it, or perhaps there may be spasm of the sphincter. A catheter can usually be passed without any difficulty and it often withdraws a large amount of urine. A large amount of urine is also passed when the bladder empties itself spontaneously in these cases. The condition is harmless and transitory.

(c) Finally, a few words are necessary in regard to those forms of pseudo-hermaphroditism which are important in the pathology of the newborn infant. Internal hermaphroditism consists of abnormalities in the internal genitals only, so that it is of no practical importance in the newborn, but external hermaphroditism always raises the question of "male or female," and it may be very difficult indeed to reply to it.

According to Neugebauer, the two forms of external hermaphroditism may be described as follows:—

(1) *External Female Pseudo-hermaphroditism*.—The clitoris is hypertrophied and may be erectile, and is sometimes perforated in its whole extent by the urethra, thus resembling a penis. The labia are more or less adherent and they resemble an empty scrotum. If a structure which feels like a testicle is palpable in one or both halves of this apparent scrotum, or at the external opening of the inguinal canal, it is easy to make a mistake in regard to the sex (misplaced ovaries). In other cases, the clitoris is hypertrophied and the vulva quite normal; or the hypertrophied clitoris may be associated with an adherent vulva, so that no vaginal orifice is visible, or there may seem to be an opening in an apparently divided scrotum. The urethral orifice, which is essentially the external opening of the urogenital canal, constitutes the common exit for the urethra and vagina. These cases exhibit a perfect resemblance to a hypospadias penisrotalis.

(2) *External Male Pseudo-hermaphroditism*.—This is the most common form. A vulva is imitated by a hypospadias penisrotalis, associated with a more or less rudimentary development of the hypospadiic penis. There are many cases wherein such a vulva appears to be so natural, that the possibility or probability of a mistake in the sex does not suggest itself. In most of these cases there is only hypospadias of the penis, or this may be combined with partial division of the scrotum and the urethral orifice in its upper half. In other cases the scrotum is completely divided, and small rudimentary labia are imitated by the edges of the cleft urethra. The internal genitals are male, corresponding to the testicles.

Neugebauer advances the following points to decide the sex: As the female appearance of the external genitals of male pseudo-hermaphrodites is due chiefly to the presence of a hypospadias, the true sex can be established by proving that the testicles, epididymis and spermatic cord exist. One cannot rely upon palpating the prostate, per rectum, in a newborn infant. This gland may indeed

be absent, but whether it is felt or not, this examination is not conclusive as to sex. If it is possible to feel definite testicles in the apparently large labia the male sex is established, notwithstanding that there may be the orifice of a vagina containing the ring of a hymen, with small labia beneath the opening of an apparently female urethra. These labia are really the edges of the cleft urethra. Klebs states that the presence of labia minora is conclusive for the female sex, but this is not the view of Neugebauer, because he has observed cases wherein there were definite testicles, although the vulva was quite of the normal female form and there was no trace of hypertrophy of the clitoris. If testicles and epididymis cannot be definitely felt in the newborn, the sex must remain doubtful until subsequent examination settles the point. Sometimes it may be impossible to decide before the age of puberty. If the testicles are concealed on both sides a decision must be unconditionally postponed, unless one is prepared to take the risk of a gross mistake. One must guard against the error, into which many practitioners fall, of assuming that an abnormally large clitoris constitutes a case of male pseudo-hermaphroditism.

Other abnormalities are often associated with pseudo-hermaphroditism, e.g., split pelvis, ectopia vesicæ, epispadias, atresia of the anus or of the urethra, spina bifida, hydrocephalus, &c. Many infants with pseudo-hermaphroditism and other abnormalities are born dead or non-viable.

CHAPTER VII. NERVOUS SYSTEM.

(1) Convulsions.

It has been customary to divide the various forms of convulsions of infancy into two classes—(1) the organic (or symptomatic), (2) the functional (or idiopathic). The former are associated with demonstrable anatomical or histological changes in the central nervous system, in the latter no such lesions have been demonstrated by our present methods of research. Despite the superficiality of such a differentiation, the separation of the two groups has proved of value from the clinical standpoint. After the third month, convulsions are more commonly of the functional type, whereas among the newly born they are usually caused by recognizable anatomical lesions; but this generalization does not always hold.

(1) Functional Convulsions.

There is one form of convulsions which stands forth prominently amongst the numerous types of functional convulsions of infants,

by reason of its clinical picture and its constant causation, viz., *tetanus neonatorum*. The cause of the disease and the path of infection are known, and despite the absence of characteristic anatomical changes we have a fairly clear conception of the pathogenesis of its symptoms. The characteristic tonic convulsive rigidity and the lock-jaw impart to it special clinical features (*vide infra*).

There is another distinct form of functional convulsions of early infancy, the etiology of which is indefinite, viz., *eclampsia neonatorum*. It should be stated here that we do not use the term "eclampsia" in connection with the newborn, in the generalized sense employed by paediatricians in describing convulsions in older infants, for they use that term to describe all varieties of convulsions (eclamptic attacks). We limit the term "eclampsia neonatorum" to convulsions occurring, within the first few days of life, in children born of mothers who have suffered from eclamptic fits during the latter period of pregnancy, or after labour. This maternal eclampsia is peculiar to pregnancy, and is associated with albuminuria and nephritis; it is characterized by convulsions and loss of consciousness, and is probably the result of an auto-intoxication by some metabolic poison which has not yet been identified. It has been shown repeatedly that the children of eclamptic mothers are in great danger from the poison circulating in the maternal blood. It is, of course, true that in very many cases these infants manifest no signs of any disease which can be connected with the maternal condition—either at birth, or later on; but there are many cases in which the injurious effect of the maternal disease is very evident (Davis). Many infants die before birth or shortly after; others are born in a very enfeebled state and die within a few days from "debility," with degeneration of the internal organs; others suffer from other diseases within the first few days, e.g., a hæmorrhagic disease. There are but very few cases which present symptoms resembling the maternal eclampsia. It is very exceptional to find that the infant has nephritis (v. p. 332); convulsions are more frequent but are rare. Esch was able to collect thirty-two cases of eclampsia neonatorum in 1910; some were his own and others were taken from the literature. The convulsions always occur, in the infant, in the first few days; sometimes a few minutes after birth, hardly ever later than the second day. The attack usually begins in the ocular muscles, cyanosis then occurs and tonic and clonic spasms of all the muscles follow. The duration of the convulsions varies from seconds to minutes; they may occur in separate attacks or follow continuously; sometimes they are abortive. There appears to be no relation between the severity of the maternal eclampsia and the development of the intensity of the infantile fits. Indeed, it appears that the infantile convulsions are likely to be more frequent in cases where the maternal eclampsia has been mild. Kreuzmann has observed a case of convulsions in an infant whose mother had suffered from nephritis during pregnancy, although she had no fits. These convulsions only affect infants who are full time or who are approaching

this period. It is doubtful whether the occurrence of convulsions in the infants of eclamptic mothers renders the prognosis any worse. Sudden death may occur during a convulsive seizure (Dienst), but in other cases there may be no symptoms of disease and the children remain well, at any rate for the first few weeks, after the cessation of the convulsions (Esch). The treatment consists of giving fluids either by the mouth or per rectum or subcutaneously, in order to wash away the poison.

Tetany or the spasmophilic diathesis is another well defined clinical condition, which occurs among the convulsive diseases of early infancy. The two terms just mentioned are often used synonymously, although logically spasmophilia indicates the general predisposition of infants to convulsive disorders, and tetany should be included in the wider term spasmophilia. The characteristic symptoms of typical tetany have hitherto not been observed in the newborn infant—either latent tetany with the triad of symptoms of Erb, Chvostek and Trousseau, or the manifest tetany with spasms of the hands and feet, tonic and clonic convulsive seizures and spasm of the larynx. The diagnosis of latent tetany is hardly possible in the first few weeks of infancy because it is very difficult to apply the electrical test of hyper-irritability. The electrical irritability of the peripheral nerves in newborn infants is easy to elicit, whereas in older infants the irritability is diminished. There are no observations which enable us to determine whether a relatively increased electrical hyper-irritability exists in the first weeks. Among Yanase's cases of parathyroid hæmorrhages there were two children aged twelve and fifteen days with anodal hyper-irritability. The youngest child with kathodal hyper-irritability was twenty-six days old. In four children, varying in age from one to nine days, with the diagnosis of "eclampsia" and parathyroid hæmorrhages, nothing abnormal was found in the nervous system. The facial phenomenon, contraction of the muscles of the face on tapping the hinder portions of the cheek, occurs frequently in newborn infants, but this cannot be regarded as pathognomonic of tetany at this early age (v. p. 73). Escherich describes a case of pseudo-tetany ("persistent form of tetany") in an infant twelve days old, but he is doubtful whether the case really comes under the classification of tetany. Apparently typical cases of tetany with laryngeal spasm do not occur within the first three months.

E. Kehrer (*Arch. f. Gynækol.*, 99, 1915, 372, and *Jahrb. f. Kinderheilk.*, 27, 1915), has recently described cases of this convulsive condition among newborn infants. There were flexor contractures of the limbs, main en griffe, positive facial and Trousseau phenomena, mechanical hyper-irritability of the muscles, and rapid improvement after administration of calcium salts. It is, however, doubtful whether one is justified in regarding these cases as tetany on the evidence of the convulsive symptoms of the illness, during which somnolence and fever occurred, but which was free from laryngeal spasm. The author recently had under his care a child with subdural hæmorrhage, which not only presented a very active facial phenomenon, but also had Trousseau's hand-posture and an extreme degree of irritability of the muscles of the limbs. Erdheim performed the post-mortem, but he was unable to find any anatomically

recognizable changes in the parathyroid glands. The tetanoid symptoms in this case were certainly due to the cerebral lesion, and it is quite possible that many cases may produce analogous effects, without the presence of the diathesis which we call tetany.

The late onset of tetany is remarkable, because its origin has been attributed to hæmorrhage in the parathyroid glands, which may occur at birth. Experimental injuries to these structures in animals have caused symptoms closely resembling tetany. Erdheim was the first to call attention to these hæmorrhages, and he regards them as related to asphyxia at birth. Yanase examined eighty-nine infants of various ages, and found in 37 per cent. of them histological traces of hæmorrhage in the parathyroids, and he considers it very probable that these hæmorrhages start at the commencement of post-fœtal life. The extent of the hæmorrhage is variable; the examination of the glands in older infants may reveal blood-cysts or only pigment residues, or all traces of the hæmorrhage may have vanished. Asphyxia is the most common cause of the bleeding; but Yanase has observed cases of typical congestive ecchymoses in the pleura and pericardium with complete freedom of the parathyroids. On the other hand, in thirty-three cases of hæmorrhage in the parathyroids, the labour was quite normal in nineteen; but asphyxia is not always the result of a protracted labour.

More recent investigations have failed to discover morbid changes in the parathyroids in cases of tetany. This circumstance, in conjunction with the fact that the clinical symptoms of the condition do not supervene for many months, has led many paediatrists to discard the view held, particularly by Escherich, that damage to the parathyroids is the cause of tetany. Although the objections to this theory may be justified, one must not forget that the absence of morbid changes in the parathyroids of older infants does not dispose of the possibility of some functional disturbance arising at the time of birth. Further, many "diatheses" have a long latent period, but we do not therefore deny that the "predisposition" to disease is congenital. If we evince no surprise that spasmophilic diathesis, of unknown etiology, makes its appearance after the third month, we need not wonder at it, if some more definite view of its etiology is maintained. We must assume that the predisposition is congenital, and that there is some exciting cause which provokes the symptoms. If we adhere to the parathyroid theory of tetany, we can explain the period of latency by assuming that there is a transference of maternal material to the fœtus, just in the same way as we explain the late onset of congenital myxœdema, by a transference of maternal thyroid secretion to the child.

Besides these clinically well-defined convulsions of a tetanoid character young infants also suffer from convulsions, which are apparently primary, and which come on in the course of various illnesses. Although the causes of these convulsions differ, the clinical picture is uniform.

It is possible that newborn infants are to a certain extent resistant to the poisons which excite these convulsions. As a matter of fact these convulsions are rare in the newborn, perhaps because the exciting causes are rare. We must not assume that there is a lesser tendency for the newborn to suffer from convulsions, because they occur very frequently when there are evident lesions of the brain. On the other hand, it is supposed that these newborn infants exhibit an increased reflex irritability, dependent upon the imperfect control of the higher cortical centres of the brain over the subcortical ones; but this theory does not correspond with the facts.

Febile conditions appear to be able to excite convulsions in young infants. Convulsions may occur in septic diseases, although the post-mortem fail to reveal any meningitis or encephalitis (Thiemich). An excess of carbonic acid in the blood possibly accounts for certain forms of convulsions. Terminal convulsions are observed, much of the same nature as those which occur in cases of temporary attacks of cyanosis during the first days of life (Snow). Nevertheless, it must be stated that convulsions are not usually associated with the attacks of cyanosis, which occur with special frequency in premature infants. Stamm noted that one child with a high temperature was attacked with convulsions after a feed of cow's milk, another child after a warm bath. Each attributes convulsions sometimes to a sudden change in the temperature of the room, from hot to cold and vice versa. But it is difficult to exclude other causes in these cases, e.g., contusions of the brain, slight lepto-meningeal hæmorrhages, &c. Occasionally, severe and persistent attacks of clonic spasms, without any other serious symptoms, are seen in infants 2 to 3 weeks old. After some time they cease absolutely. Possibly these convulsions are the transitory results of some slight brain lesion.

Epilepsy sometimes dates back to early infancy, appearing either as a complication of one of the infantile cerebral paralyses or as the idiopathic form. Many cases which come on later in life, as functional nerve disease, are to be attributed to some sequela of a cerebral lesion sustained at birth, especially meningeal hæmorrhage (Neurath).

Zipperling describes a form of motor irritability which is hardly recognized by medical men, but which is quite familiar to midwives—indeed, 40 per cent. of newborn infants suffer from it. It affects the ocular muscles and the facial region; the rest of the body is free. Zipperling describes the symptoms as follows: The eyes become suddenly deviated, or conjugated, or turned in every conceivable position; associated with these movements is a brief spasm of the lids and very rapid contractions of the entire orbicularis oculi muscle. Many infants also exhibit some drawing-up of the angle of the mouth lasting for a few seconds. This constitutes the entire attack. The infant sometimes draws a deep breath and becomes quiet; but the process suddenly starts again. The eyeballs are often in active rotatory movement under the closed lids. The attacks may last for a few minutes, and be repeated very frequently—as much as one hundred times in the day. After a few

months they apparently vanish. The symptoms are usually so slight that they escape detection, unless close observation is maintained. The "smile" of the infant in the very early days is possibly a manifestation of this irritability. Zipperling thinks that the phenomenon is physiological; he considers that it is caused by circulatory disturbances in the area of origin of the nuclei of certain motor cranial nerves.

(2) Convulsions of Organic Origin.

The following conditions are liable to cause convulsions: Intracranial injuries to the meninges or brain during labour; inflammatory diseases of the cerebral cortex or meninges; disturbances in the development of the cortex and their sequelae; hydrocephalus. Further details are given under the appropriate headings. The symptomatic treatment of convulsions has been discussed earlier in this work (v. p. 217).

(II) Organic Diseases of the Central Nervous System.

(1) Disturbances in Growth and Deformities.

ANENCEPHALY.

Anencephaly means the congenital absence of the cerebral hemispheres. They are either completely missing or are replaced by a vascular mass. As a rule the medulla and spinal cord and some of the basal ganglia are properly developed; the cerebellum is usually atrophic. It is rare to find an absence of the spinal cord associated with anencephaly. Children with these deformities are not viable.

The vault of the skull is usually absent in anencephaly. The skull of the cerebellum appears to be replaced by a red spongy material. Very vascular fibrous tissue is found under a skin-like integument. The deformity has been assumed to originate in a foetal meningitis or in some arrest of development, and has been associated with the following factors: Premature cleft formation, imperfect closure of the medullary tube, deficient deposit of ectoderm, amniotic adhesions and toxic conditions of a chemical nature. It has been stated that aplasia or hypoplasia of the adrenal glands has been found in some cases of anencephaly. This has given rise to the not very probable suggestion—that adrenal inadequacy is responsible for the deformity. Possibly there is some hereditary influence at work, for anencephaly occasionally runs in families. It is very often associated with other deformities.

An anencephalic infant may survive for a few days. The longest duration of life hitherto observed has been sixteen days (Ziehen). The infants are able to swallow and make chewing movements, to cry and to respond to irritation of the skin. The muscles are usually in a state of great hypertonicity. The muscular reflexes are in-

creased; sometimes a coarse tremor exists, sometimes there are definite convulsions. The action of the bowels and the bladder is normal. Mimetic reflexes are stated to occur, on the application of bitter or sweet substances to the tongue. Occasionally reflexes are produced by the irritation of a strong light (Heubner, Wichura, Sternberg, Latzko).

ENCEPHALOCELE.

Encephalocèle means a prolapse of a portion of the brain together with the meninges. *Meningocèle* means a prolapse of the dura mater and pia-arachnoid through a pre-existing opening in the skull. *Hydro-encephalocèle* or *encephalo-cystocèle* means the prolapse of a portion of the brain which contains a cavity corresponding to a ventricle. Encephalocèles and meningocèles do not usually alter in size after birth; but a hydro-encephalocèle undergoes a rapid growth.

Three main forms of encephalocèle are to be distinguished (Ziehen).

(1) *Basal encephalocèle*.—The hernia appears in the nasal or pharyngeal cavity. An intra-nasal encephalocèle, wherein a portion of the frontal lobe projects into the nasal cavity via the lamina cribrosa of the ethmoid bone, obstructs nasal respiration, and sometimes appears as a tumour extruding from the nasal orifice (E. v. Meyer). A spheno-pharyngeal encephalocèle appears between the sphenoid and ethmoid bones. Exner describes a case of basal encephalocèle wherein a cyst protruded into the mouth cavity through an opening in the bony base of the skull and a cleft in the soft palate. This cyst communicated with the dilated anterior horn of the right lateral ventricle. In a spheno-orbital encephalocèle, the hernia appears through the superior orbital fissure; in a spheno-maxillary encephalocèle it appears through the inferior orbital fissure.

(2) *Frontal encephalocèle*.—The most frequent example of this group is the naso-frontal variety, wherein the hernia appears between the frontal bone and the nasal process. Much rarer varieties are the naso-orbital encephalocèle at the internal canthus (hernial opening between ethmoid, frontal and lacrymal bones) and the naso-ethmoidal encephalocèle at the lower edge of the nasal bone between the bony and cartilaginous portion of the nose (hernial opening at the foramen cecum).

(3) The occipital encephalocèle is the most common form of *hernia cerebri*. It is situated at the nape of the neck, below or above the external occipital protuberance (fig. 54).

The ætiology of encephalocèle has not been clearly established. Its localization is against the assumption that it occurs through the existence of pre-formed bone defect. Ziehen finds a satisfactory explanation in Geoffroy St. Hilaire's theory, according to which the *hernia cerebri* occurs through circumscribed adhesions of the rudimentary brain with the membranes of the ovum. In other forms

possibly the deficient closure of the cranium may play an important part. This is more than probable, if at the same time there are deficiencies in the region of the vertebral column.

The encephaloceles are of various sizes—from that of a walnut to that of a child's head. The skin over the tumour is either normal or thinned and shiny, sometimes of a bluish colour, occasionally

it shows scar changes, remains of amniotic adhesions. The tumours have sometimes a narrow pedicle, and sometimes a broad base. They are liable to be compressed, so that symptoms of cerebral compression may occur. Hydro-encephalocele manifests fluctuation and transparency; protrusion during screaming; straining and coughing; sometimes on change of position the swelling increases or diminishes. Some forms of encephalocele show pulsation; movement of the tumour with respiration is seldom to be seen.



FIG. 54.—Occipital encephalocele.

Large *hernia cerebri* may form an obstruction to birth. Otherwise the clinical symptoms are, as a rule, not remarkable at this early

period. These symptoms vary according to the seat and size of the hernia, to the quality of the protruded portion of the brain. Whereas a small *hernia cerebri* may run its course without further symptoms, the rapidly enlarging forms offer a very poor prognosis. Left to themselves, hydro-encephaloceles often burst, or may result in severe loss of function, idiocy, paralysis, &c. With large tumours the children often succumb to secondary infection.

Treatment is essentially surgical. The former practice of puncture and compression has given way to the radical removal of the tumour (Bergmann). Those encephaloceles most adaptable to operation are those with a narrow pedicle and narrow communication, also all singipital and occipital tumours. The presence of cerebral substance and communication with a lateral ventricle present no contra-indication to the operation. This only exists with large occipital protrusions and in such cases where hydrocephalus is combined with other fatal malformations. Operations have often been performed with good results during the first few days or weeks. But relatively frequently operations at first successful have been followed by secondary hydrocephalus (Lindfors, Vogel).

Even if this is not always the case, it must be taken into consideration that cases have been known to reach a life age without any operation. Therefore, early operation should only be undertaken with rapidly enlarging tumours.

SPINA BIFIDA.

Spina bifida occurs by the union of the embryonic medullary tube failing at one place. With the so-called open form of spina bifida (rachischisis) there is in the middle of the back an open defect, at the margin of which skin, fascia, muscle, bone, dura and pia suddenly cease, and remnants of the spinal cord can be seen at the bottom. The malformation is generally combined with other severe anomalies, and is of no clinical interest owing to the non-viability of the child.

In the closed form of spina bifida (spina bifida cystica) the fissure of the vertebral column is covered through a portion of the contents of the vertebral canal being forced out like a cyst owing to pressure of collected spinal fluid, so that these formations, or part of them, lie free on the surface, or may be totally covered by external skin. Spina bifida cystica is generally situated at the dorso-lumbar or lumbosacral part of the vertebral column, more rarely at the cervical part. Three forms may be distinguished:—

(1) *Myelocoele*.—The deficiency involves skin, muscle, bones, dura, pia and spinal cord substance. The central canal is not closed. The ventral portion of the spinal cord covers the collected fluid in the anachnoidal tissue, and is forced forward as a cyst. The myelocoele is nearly always seated in the lumbar region. It forms a dark red, moist tumour, varying in size from that of a nut to an apple, fungiform, round or oval-shaped, fluctuating, mostly tightly stretched, in which several areas can be distinguished. The zona medullo-vasculosa, which corresponds to the remains of the protruded spinal cord, forms the central section; in fresh cases the entrance and exit end of the central canal can be probed and fluid evacuated. To this zone is attached the pearl-grey, delicate vascular zona epithelo-serosa; it corresponds to the turned-down pia, which is covered with epithelium which has proliferated from the margins of skin. The external surroundings form the zona dermatica which is formed of the thick skin, generally covered with hair and rich in vessels. If infective processes develop soon after birth on the oozing base of the innermost zone, or the cyst becomes ruptured during birth or bruised or lacerated, the anatomical form loses its clearness.

(2) *Myelocystocoele* is an enlargement of the central canal and a protrusion of the dorsal wall of the spinal cord. The external skin coverings are closed.

(3) *Meningocoele*, very rare in its pure form, is due to a protrusion of the lepto-meninges on the dorsal aspect of the intact spinal cord. It is generally seated over the sacrum. When this meningocele is situated on the ventral surface of the bone the term used is *spina bifida anterior*.

(4) *Spina bifida occulta*.—In the absence of an accumulation of fluid, in this case there is no protrusion. But in spite of this the cord and nerve roots may be seriously injured.

The numerous grooves and sinus-like depressions and clefts in this region constitute the most rudimentary forms of spina bifida. They occur in about one-third of all newborn infants in an area of 1.33 cm. from the anus (Markoe and Schley). They may indicate a "myelodysplasia," but as a rule they possess no pathological significance.

The clinical symptoms of spina bifida depend upon the involvement of the spinal cord. In myelocoele and myelocystocele there is usually a paraplegia or a paresis of both lower limbs, particularly the legs, also paralysis of the sphincters with incontinence of urine and faeces, prolapse of the anus and disturbances of sensation. Meningocele does not necessarily cause any nerve derangements.

There is not usually any difficulty in diagnosis. In the absence of any paralytic symptoms a meningocele may be mistaken for a sacral tumour, but sacral teratomata are harder and nodular. A typical myelocoele, the most frequent form of spina bifida, cannot possibly be mistaken.

The prognosis of spina bifida is bad; the most favourable variety is that wherein the cyst is covered by skin. The children usually die from infection of the swelling or from cystitis and pyelitis, though the fatal termination does not usually occur in the newborn period. If the parts are kept scrupulously aseptic, infection may be avoided. If the moist tumour is daily powdered with dermatol and covered with sterile gauze, it may gradually acquire an investment of skin and become headed over; but the functions which have been destroyed cannot be recovered. Hydrocephalus very often develops later in these cases. An equally unfavourable prognosis follows all operative measures (puncture with subsequent injection of such irritating substances as iodine; injection of paraffin; excision of the sac and reduction of the portion of the spinal cord displaced into it). It is very doubtful whether operation is of any real benefit to the infant, even if it is successful. Even if there be no paralysis and the swelling is covered by skin, the danger of operation is so great that the decision to resort to it should not be made lightly.

APLASIA AND DYSPLASIA IN THE REGION OF THE CEREBRUM.

There is a group of diseases of the central nervous system, due to developmental defects and derangements. These diseases may affect the cortex in the motor area, or the nuclei of the cranial nerves. According to Ziehen, the cortical and nuclear aplasias and dysplasias may be classified as follows—unless they are caused by disturbances which supervene at a later age of infancy:—

(1) Simple defects in development (simple aplasia and hypoplasia).

(2) Intra-uterine destructive processes, with corresponding disturbance of development (intra-uterine dysplasia).

(3) Destructive processes during birth, with corresponding disturbance of development (natal dysplasia).

Cases, wherein normal development has been interrupted and delayed by premature birth, must be classified in the first group. In contrast to hypoplasia and aplasia, these forms manifest a tendency to involution and retrogressive development, in the narrow sense, and therefore may be regarded as a hyosteroplasia. The second group includes infective and inflammatory or traumatic and hæmorrhagic processes in the grey matter or destructive processes in the cerebral cortex and pyramidal tracts, resulting from intra-uterine meningitis. The ætiology of these fetal inflammations is unknown; the only cause which is definitely responsible for these conditions is congenital syphilis. Injury to the gravid uterus may possibly play some part. Injury at birth and asphyxia are causes of natal dysplasia; possibly also the traumatic effect of attempts at resuscitation.

The clinical pictures of these congenital diseases are represented in the case of the nuclear form by the so-called "infantile nuclear atrophy," and in the cortical form by the congenital cerebral palsies of infancy.

(a) Congenital Cerebral Palsies ("Infantile Nuclear Atrophy").

The congenital paralyses are most frequently manifested in the ocular muscles, the levator palpebræ being the one generally affected (congenital ptosis), but the other muscles supplied by the oculo-motor or abducens nerve may also be involved. Complete ophthalmoplegia externa is very rare. The paralyses are often symmetrical, an important point in the differential diagnosis from traumatic paralyses. The condition is often associated with other cranial nerve paralyses, and also with various abnormalities in development (absence of fingers, defects in muscles, changes in the auricular cartilage, &c.) (Bernhardt, Schmidt). It is hardly possible to make an accurate analysis of the clinical picture in the early days of life.

Derangements in development in the area of the facial nerve are more rare. The paralyses may be unilateral or bilateral; they may affect the upper or lower branches or both branches. It is easy to confuse this condition with the harmless traumatic peripheral facial palsy, and with the cortical paralyses which may follow injury during labour. The diagnosis becomes quite obvious in the course of a few days, for peripheral paralysis recovers rapidly, while other focal symptoms appear in the case of cortical paralysis.

Congenital bulbar paralyses are extremely rare, if indeed they occur at all.

The occurrence of a congenital nuclear aplasia is firmly established (Moebius, Heubner, &c.); but nevertheless anatomical investigations in a large number of cases of congenital paralyses in the region of the ocular and facial muscles have shown that there is no nuclear disease, but merely lesions in the nerves or muscles. The differential diagnosis of these paralyses is hardly possible during life. In unilateral defects of the ocular movements and in unilateral facial paralysis, the lesion is almost always peripheral (Zappert).

The prognosis of congenital paralysis of cranial nerves, whether the lesion be nuclear or peripheral, is absolutely unfavourable as far as recovery from the symptoms is concerned. In cases of intra-uterine dysplasia there is some possibility of improvement; but it is obviously impossible to distinguish these *a priori*.

(b) The Congenital Forms of Cerebral Infantile Paralysis.

It is necessary to distinguish those cases wherein the cerebral paralysis already exists at the time of birth, from those wherein the typical symptoms appear to indicate that an injury to the brain has occurred during labour. This latter class has already been discussed in connection with intra-cranial injuries. It has been stated that the paralysis which results from a dural hematoma and brain injury is usually of the hemiplegic type, whereas spastic diplegia (Little's disease) is associated with bilateral lepto-meningeal or cortical haemorrhages and other lesions. The comparative frequency of the latter type among premature infants is probably due to the fact that the motor tracts are incompletely developed and therefore more susceptible to injury. But the cause may be merely an interruption in the normal development of the motor areas and the pyramidal tracts, without the influence of any injury, i.e., a dyserogenesis in the sense previously indicated. Infants, affected in this way, may be expected to exhibit a well-marked picture of paraplegic rigidity in the very early days of life. Intra-uterine dysplasia in the cerebral cortical area, following upon fetal meningitis, leads to paraplegia and hemiplegia which are evident immediately after birth; later on it is the cause of defective intelligence and epilepsy, &c. It is often impossible to differentiate clinically between the traumatic and non-traumatic forms.

(2) Inflammatory Diseases of the Brain.

(A) ENCEPHALITIS.

Encephalitis of the newborn is a subject which is very obscure from the clinical, aetiological and pathological aspects.

Vinchow, years ago, described *congenital interstitial encephalitis* as consisting of changes in the grey matter of the cerebrum caused by a diffuse or localized infiltration of fatty granular cells. Later on, Jastrowitz and Limbeck stated that this condition was not pathological. The histological investigations of Merzbacher suggest that there may be both "physiological" and "pathological" granular cells; but we need not now dwell upon the significance of this point. No one has yet described a clinical picture to accord with an encephalitis of this character.

Brain defects (parencephaly) have been attributed to congenital encephalitis. Leitz maintains that they are due partly to the sequelae of hemorrhages, and partly to changes acquired in extra-uterine life and caused by infection or by toxæmia.

Suppurative encephalitis is the least obscure of the varieties,

because it is clearly due to infection which has occurred before or after birth.

The author observed the case of a premature infant, weighing 2,350 gm., delivered by breech owing to the comparatively large circumference of the head (36 cm., the length of the body being 45 cm.). The infant's head was remarkably asymmetrical towards the right side; there was facial paresis of the left side and spasm of the left upper extremity. Temperature, 102.2° F. The infant survived two days. At the autopsy there was found encephalitis of the right cerebral hemisphere, with pyocephalus of the right side and dilatation of the lateral ventricle.

Septic encephalitis occurs as a metastatic condition or as a meningo-encephalitis. The morbid anatomy of the former consists of multiple foci in the grey substance of leucocytes and bacteria (Fischl). The latter arises as an extension of purulent meningitis into the cerebral tissue. It is almost impossible to recognize these conditions during life, more especially to differentiate between encephalitis and meningo-encephalitis.

Feuchtwanger reports the case of an infant who was quite well until the fifth day of life, when transitory pyrexia and cyanosis supervened. Later on, this child suffered from imbecility and convulsions. He regards this case as one of polioencephalitis.

Syphilitic meningo-encephalitis will be discussed subsequently.

(B) MENINGITIS.

The cause of foetal meningitis, which is associated with developmental disturbances in the cerebral cortex, is still quite unknown. The condition may apparently be acute or chronic. We also know very little about the nature of the serous meningitis, which we must assume is the cause of congenital hydrocephalus.

Pachymeningitis interna haemorrhagica probably occurs only in infants affected with hereditary syphilis.

Purulent meningitis is caused by an extension to the meninges of suppurative inflammation of the middle ear or naso-pharyngeal space, or it may arise by metastasis. Sometimes the symptoms are typically meningeal, viz., convulsions, cervical rigidity, bulging of the fontanelles and sutures, &c.; at other times the symptoms are suggestive of tetanus (de Bruin). But the disease may run its course without any characteristic symptoms. The infants simply waste, they are weary, suck feebly, and the post-mortem reveals that the cause of the "debility" was purulent meningitis. The diagnosis might be made by lumbar puncture, but as the disease is so rare, the possibility of meningitis is not thought of until the appearance of symptoms of cerebral irritation. It is remarkable that meningitis may run its entire course in the newborn without any rise in temperature, or pyrexia may not appear until towards its termination.

The prognosis is hopeless. The disease may be fatal in twenty-four hours; but may last as long as eight to fourteen days. As the symptoms are indefinite, it is not always certain when the disease actually commenced.

The following organisms have hitherto been incriminated as the causes of purulent meningitis: *B. coli* (Hlisdale, Goldreich, personal case), *B. lactis aerogenes* (Scheib), *B. vulgare capulatus* allied to *Friedländer's bacillus* (Bonhoff and Esch), *B. pyrogenus* (Benley).

The phlebotic form of sinus thrombosis, which is observed after septic diseases in the vicinity of a sinus, occurs occasionally in newborn infants, e.g., after aurial or nasal suppuration. It may possibly also arise through blood infection in general pyæmia. Hamill describes a case of extensive sinus thrombosis in an infant aged seven days, who was suffering from umbilical sepsis. The symptoms which may be anticipated in sinus thrombosis consist of extreme fullness of the cutaneous veins and œdema of varying localization (according to the position of the affected sinus); bulging of the fontanelle, &c.; but no case has yet been diagnosed in the newborn. It is almost impossible to differentiate it from purulent meningitis.

(C) CONGENITAL HYDROCEPHALUS.

This term is applied to the condition of serous effusion into the cerebral ventricles with consequent distension (congenital internal hydrocephalus), or to serous effusion on the surface of the brain (congenital external hydrocephalus). The latter form is the more rare, but it does occur in the newborn (Bókay), sometimes in association with meningocæle (Hammerschlag).

Internal hydrocephalus is meant when congenital hydrocephalus is spoken of without qualification. It represents a transudation or an exudation. If there be any obstruction to the outflow of the cerebrospinal fluid, such as may occur after an intra-cranial hæmorrhage, we may assume that transudation is the cause. (Vide p. 246.) Jonkowsky records a case of hydrocephalus in a newborn infant who had a cyst in the pineal gland. Probably, congenital internal hydrocephalus is generally preceded by an inflammatory process, an intra-uterine serous meningitis or meningo-encephalitis, the cause of which is very obscure. Syphilis is not, as a rule, the cause of congenital hydrocephalus. Possibly alcoholism in the parents and injuries to the pregnant uterus may play a part in the causation; but the main cause is undoubtedly some infective process of unknown origin, which is transferred from the mother to the foetus.

The inflammatory process which causes the hydrocephalus may have ceased at the time of birth, but it usually continues after birth. Most cases of congenital hydrocephalus increase in size after birth, and many do not become evident until some time after birth. If the accumulation of fluid starts early in intra-uterine life, the development of the brain may be seriously arrested. The external circumference of the skull is not necessarily enlarged, indeed it may be abnormally small and present the characteristic shape of micro-

cephaly. In such cases, the whole brain is converted into a watery cyst (Zappert and Hirschmann), and the condition is termed *Hydro-microcephaly* or *Hydro-anecephaly* as the brain may be almost entirely absent.

As a rule, however, the skull is enlarged if there is effusion already at the time of birth, and it may constitute an obstruction to delivery, necessitating perforation or puncture of the skull. It occasionally happens that the infant lives even after an intra-partum puncture of a hydrocephalus, but, of course, this is very exceptional. The circumference of the skull may be as much as 50 cm. at birth, and may present the characteristic shape of a hydrocephalus: an enlargement of the vault of the skull out of proportion to the bony face, protrusion of the frontal and parietal bones, steepness of the forehead or forward inclination thereof, outward inclination of the squamous portion of the temporal bones, great widening of the sutures of the skull and enlargement of the fontanelles, altered position of the eyeballs, &c. The diagnosis is easy in such cases, even if there be no marked tension or bulging of the membranous part of the skull, which may be the case if the process is arrested. The other symptoms of hydrocephalus may also be detected in the newborn, viz., hypertonicity and spasm of the muscles, increase of the reflexes, convulsions, psychical disturbances such as apathy and disinclination to take nourishment.

The diagnosis is more difficult if there be no enlargement of the skull, or if its shape be not characteristic. A widening of the sutures and an abnormal enlargement of the fontanelles is not necessarily the result of hydrocephalus if there be no extreme tension; a bulging of the membranous portions of the skull may be the result merely of an intracranial hæmorrhage or meningitis. In such cases the diagnosis may be determined by means of lumbar puncture or by transillumination of the skull.

The prognosis is difficult during the early days of life, because the course of the disease is uncertain. We can only tell whether the process is stationary or progressive by the behaviour of the intracranial pressure. But as it is normal for an infant to lose fluid from the body during the first few days of life, and as this causes a decrease of tension in the fontanelles the inferences from changes in the intracranial pressure are liable to be erroneous.

Lumbar puncture is the only treatment which can be entertained during the first few weeks; although this procedure may be only symptomatic treatment it may be employed in the very earliest stages.

(3) Diseases of the Spinal Cord.

Congenital myatonia (Oppenheim) (*minia musculorum congenita* of Töbler, *myohypotonia* of Bernhardt, *amyotonia congenita* of Griffith) consists of congenital weakness in the muscles of both legs. The arms are also sometimes affected, and occasionally the muscles of the trunk and neck; the region supplied by the cranial nerves is very rarely involved (Pollak). The paralysis is not usually

complete and some power of active movement still remains. The disease is never progressive; the paralysis usually decreases to a considerable extent in the course of months, but its subsidence is very slow and no case of complete recovery has hitherto been observed. The prognosis as far as life is concerned is usually good, but if the respiratory muscles are affected the children are very liable to acute pulmonary disease, to which they frequently succumb. These cases must be distinguished from deformities and injuries during birth, in the vicinity of the vertebral canal; the latter cause paralysis of the sphincters as well as the paraplegia.

The disease begins during fetal life. Tobler states that the intra-uterine movements are less active than usual. Neurologists are not agreed about the pathology of the disease. The primary seat of the disease is partly in the muscles, partly in the peripheral nerves and partly in the spinal cord (Archangelsky and Abrikosoff, Bernhardt). Histological examination shows that it is either a form of spinal muscular atrophy (Rothmann) allied to the familial muscular atrophy (Werdnig-Hoffmann) or a form of fetal poliomyelitis (Marburg).

Comby describes a case of *chronic spinal muscular atrophy*. From birth there was persistent diffuse paralysis of the muscles of all the limbs, of the back and of the intercostals. The child died at the age of five months from asphyxial seizures.

Acute poliomyelitis, as an acquired disease, is very rare during the newborn period. Although recent years have witnessed numerous epidemics of this disease, only one case is recorded as occurring in an infant fifteen days old. There was no antecedent pyrexia, but there was paralysis of the extremities, which cleared up completely after four weeks (Falk). But there is no real evidence that this was a case of poliomyelitis.

(III) Diseases of the Peripheral Nerves.

The most important of these consist of the paralyses which result from difficulties in labour, in the region of the brachial plexus and the facial nerve (v. p. 182). Other forms of paralysis are of less importance because of their rarity.

Congenital musculo-spinal paralysis shows itself by a drooping posture of the hand, in consequence of the extensor paralysis; sometimes this is accompanied by a secondary contracture of the flexor muscles. Bonnaire found congenital atrophy of the nerve in such a case. Spieler observed in an infant who had congenital musculo-spinal paralysis, that there was a brownish red patch of discoloration below the elbow which later on developed into an ulcer, suggesting that an amniotic band had constricted the forearm in the uterus. Kirnsson also reports a case of clubbed hand which he attributes to the same cause. This etiology is convincing when a circular furrow is found constricting the limb in the position where the musculo-spinal nerve winds round the humerus (Cassirer).

Joachimsthal and Pettessohn have described the analogous type of paralysis of the peroneus leading to the development of a club-foot. The furrow from the constricting band was found either at the upper end of the leg or at the junction between the lower and middle third.

The prognosis in regard to the recovery of these paralyses is unfavourable. Permanent limitation of movement and disturbances of growth usually result. The treatment is by orthopaedic methods, but the question of liberating the nerve from any remaining constriction may arise.

CHAPTER VIII.

EYE AND EAR.

(A) Diseases of the Eye.

(i) Congenital Diseases.

Congenital diseases of the eyes may be divided into several classes from the aetiological standpoint (Leitner). One class depends upon maldevelopment or defective development. Maldevelopment includes conditions wherein the causes, which produce inflammation, participate, and also those which result from some traumatic effect of the amnion. Well developed healthy eyes may suffer damage from intra-uterine inflammation. There are many congenital diseases of the eyes, but we will only refer here to those which are clinically manifest in the newborn infant and which come within the purview of the general practitioner, for prognosis and treatment.

(A) LIDS AND LACRIMAL DUCTS.

Congenital coloboma of the lid appears as an acute angled or circular indentation in the upper lid, or more rarely in the lower. It is often associated with changes in the surrounding parts, the eyeball, face, mouth, &c. A coloboma of the lid may be remedied later on, by a plastic operation, in accordance with the degree of the abnormality.

The so-called mongolian or epicanthic fold is usually quite recognizable in mongols, soon after birth.

The following congenital abnormalities of the lids are more rare: Entropion in congenital facial hemiatrophy, atrophy of the skin of the lid in microphthalmia, &c.; symblepharon (adhesions between lid and eyeball), ankyloblepharon (adhesions between the margins of the two lids), &c.

An absence of the cleft between the two lids is called cryptophthalmos. This abnormality is due to the fact that the mesoderm and ectoderm covering the primitive ocular vesicle have not developed

into cornea, but into skin. The development of the lids and conjunctival sac remains in abeyance. Operative interference is impossible owing to the rudimentary development of the eyeball. The abnormality may be bilateral (Goldzieher).

The congenital tumours of the lids and their vicinity include dermoids, naevi, angiomas and lymphangiomas, fibromata, neuro-fibromata and lipomata. The proper time for operating depends upon the size of the growth, and it is quite unnecessary to interfere too early, for fibrous degeneration and spontaneous diminution may occur, especially in the case of angiomas.

Among the various abnormalities of the lacrimal apparatus, congenital occlusion of the lacrimal duct is of most practical importance, because this may lead to inflammatory symptoms in consequence of retention of the contents of the lacrimal sac. Atresia and absence of the puncta lacrymalia, and congenital fistula of the lacrimal sac have been described.

(B) CONJUNCTIVA, CORNEA AND SCLEROTIC.

Congenital abnormalities of the conjunctiva and sclerotic are rare. Angiomas, naevi, dermoids, lipo-dermoids of the conjunctiva are known; the sclerotic may be congenitally thin, which is recognized by a bluish tint of the eyeball, and it may also contain scleral cysts.

The cornea is frequently the site of congenital opacities. Some are due to injuries during birth; but apart from this, most of them are caused by scars of an intra-uterine keratitis (Seefelder). They may be the result of syphilitic uero-keratitis in the fœtus. Tongue-shaped opacities at the border of the cornea and parenchymatous opacities in various situations, as well as congenital leucomata and staphylomata of the cornea have been observed. Corneal opacities often occur in association with hydrocephalus. Peters has described a form of congenital, parenchymatous orbicular opacity of the middle of the cornea with central defective development of Descemet's membrane, which is almost always bilateral. This form may also be hereditary and familial, and may be associated with other maldevelopments, such as smallness of the eyeball, persistence of the foetal pectinate ligament, coloboma of the iris, embryotoxon (abnormally extensive encroachment of the episcleral tissue over the limbus as a ring-shaped opacity of the margin of the cornea), cone-shaped cornea, hydrophthalmos. The opacity may clear up, starting at the border, and completely disappear, but it may also persist. The prognosis of the opacities previously mentioned is doubtful as far as clearing up is concerned; in the case of leucomata and staphylomata it is naturally unfavourable.

The so-called epibulbar tumours (dermoids and lipo-dermoids) are rather frequent. They are usually situated on the outer or lower section of the corneo-scleral margin, and appear as flat, semi-circular or lentil-shaped elevations of whitish or yellowish colour (Biguiss).

(C) CONGENITAL ABNORMALITIES OF THE INTERNAL EYE.

The most important abnormalities of the iris are aniridia or iridodermia, congenital complete or partial absence of the iris (frequently observed as a hereditary deformity), and coloboma of the iris. Typical coloboma appears as a vertical pear-shaped or oval cleft in the lower section of the iris; it is often associated with other colobomata of the eyeball. Atypical coloboma consists of a mere displacement of the pupil (korektropia). Congenital synechiae, and retrogression of the pigment layer of the iris are rare abnormalities.

Remains of the pupillary membrane constitute a very frequent abnormality in the form of dots and threads on the anterior capsule of the lens. Sometimes they encroach upon the pupil; occasionally the whole membrane apparently persists, resembling a film of exudation.

In regard to congenital abnormalities of the lens, opacities may occur in certain circumstances in the newborn, whereas displacements do not usually occur until much later. The different varieties of congenital cataract (nuclear, spindle, anterior and posterior polar, lamellar or complete) are often combined with other developmental disturbances of the eye (microphthalmos, &c.) and of other parts of the body. Congenital cataract should be operated on as early as possible, even before the termination of the first year.

Congenital changes in the hinder portion of the eye, such as coloboma of the choroid, a persistent hyaloid artery, exudation in the vitreous, abnormalities in the optic disc, &c., can only be detected by the ophthalmoscope. Clinical symptoms do not appear until later on. Changes in the fundus are discussed in connection with congenital syphilis.

(D) ABNORMALITIES OF THE GLOBE.

Unusual smallness of the eyeball (microphthalmos) is seen as a simple abnormality, without any other change in the eye, or it may be associated with various deformities, especially coloboma.

Complete absence or rudimentary development of one or both eyes is termed anophthalmos, and it is manifested clinically by the remarkable narrowness of the palpebral fissure, and the smallness of the orbit. Sometimes the eyeball is replaced by cysts of the lids or of the orbit. A rare deformity consists of one central orbit containing one eye, usually misshapen (cyclopia). Infants affected in this way are not usually viable.

Megalo-ophthalmos or megalocornes are the terms given respectively to abnormal enlargement of the eyeball or of the cornea, the rest of the eye being normal. This must be differentiated from another form of enlargement of the globe, the so-called congenital hydrophthalmos or congenital glaucoma, which is more important clinically. This condition is not usually sufficiently advanced in newborn infants to permit of recognition.

Exophthalmos and other displacements of the globe are the results of orbital hæmorrhages (Burkard), of dermoids of the orbit, of maxillary sinus suppuration, or of an abnormal smallness of the orbital cavity (Bertram). Congenital enophthalmos is associated with microphthalmos, or is the result of disease of the ocular muscles.

Congenital paralysis of the ocular muscles has already been referred to (vide pp. 220 and 351).

(2) Diseases acquired during or after Birth.

HEMORRHAGES.

If newborn infants are examined with the ophthalmoscope, marked congestion of the retinal vessels will often be detected, immediately after birth. The optic disc is frequently dark grey or bluish-grey in colour (Jäger, Königstein, Ulrich, Stumpf and Sacherer); at the same time, a reddish border is often seen in the position of the sclerotic ring, whence delicate flame-shaped hæmorrhagic streaks radiate. The peculiar ring of the optic disc is no doubt due to hæmorrhages within the sheath of the optic nerve, and it disappears completely after a few days (Sacherer).

Retinal hæmorrhages are found with comparative frequency. They were present in 23 per cent. of Montalchini's cases, and in 21 per cent. of Sacherer's, and in half the number they were bilateral. The hæmorrhages are round or flame-shaped; they are either scanty or may be so abundant over the entire fundus that the borders of the disc may appear to be obliterated. They begin to subside in a few days, even if they are extensive, and completely vanish at the end of six days. The small peripheral hæmorrhages are of no importance; but the larger and more deeply situated hæmorrhages and those in the macular region may injure the delicate retinal elements and possibly affect vision later. They may account for some of the cases of "amblyopia without physical signs" (Sacherer). Retinal hæmorrhages may possibly lead to the development of gliomata (Wehrli).

These retinal hæmorrhages are most probably caused by the process of birth, like the caput succedaneum. In the first position of the vertex the right eye is alone affected, or at any rate suffers more; in the second position the left eye is involved. This circumstance proves that the cause of the retinal hæmorrhages is interference with the circulation in a definite limited area of the head and the consequent congestion—brought about by the mechanism of labour. The asphyxia contributes to the causation. Infants with soft heads seem to have a predisposition to these hæmorrhages, and also premature delicate children with easily torn vessels. The condition bears no relation to the genuine hæmorrhagic diseases (Stumpf and Sacherer).

Conjunctival hæmorrhages, which are very frequent, arise in the same way as retinal hæmorrhages. They often surround the outer

half of the cornea like a crescent; but they disappear spontaneously during the first week.

Injuries to the eye during labour have already been referred to (v. p. 220).

GONORRHOEAL CONJUNCTIVITIS.

From the point of view of frequency and of possible consequences, gonorrhoeal conjunctivitis is by far the most important of the inflammatory diseases of the eye which occur during the first few days of life. Indeed it is one of the most important of all diseases of the newborn infant. Appropriate prophylaxis and timely treatment, thoroughly carried out, can prevent or cure the disease in most cases; but its neglect often leads to blindness.

Infection usually occurs when the head is passing through the vagina of a mother who is suffering from acute gonorrhoea, or who harbours gonococci in the vulva, vagina or cervical canal as a result of an attack of gonorrhoea. The whole course of the disease in the mother may be quite latent. The contagium of gonorrhoea is extremely tenacious—an infant may be infected years after the infection of the father. The longer the head is detained in the lower parturient canal and remains in contact with the infective secretion, the more likely is the infant to become infected. In protracted labours, in children with large heads, in contracted pelvis especially in primiparae, in face and occipito-posterior presentations, and in forceps cases wherein the eyelids are forced open, the risk of infection is greater. As a rule, the infant is infected at the moment the eyes are opened for the first time, and the discharge which clings to the lids penetrates to the conjunctival sac.

Occasionally, infection occurs before the actual birth, i.e., is intra-uterine. Many cases have been described, wherein infants have been born with symptoms of blenorrhoea, or wherein they have supervened by the third day before the completion of the incubation period (Hausmann, Magnus, Krukenberg, Keller, Feis, Hirschberg, Naumann, Holzbach, Dorland, &c.). In nearly all these cases, there has been premature rupture of the membranes, often several days before the onset of labour pains. After rupture of the membranes, the amniotic fluid becomes infected, and after penetrating through the palpebral fissure, gains access to the eyes. These are especially liable to be affected, in the infected amniotic fluid, by the gonococci; the process is very rapid with the high temperature which prevails in the uterus. Gonorrhoeal ophthalmia, acquired in the uterus, is usually very severe, and rapidly causes disease of the cornea; sometimes a corneal ulcer is already present when the child is born.

The infection may also take place *after* birth—an event which is not at all uncommon. If symptoms do not appear until after the fourth day, the case may be assumed to come within this category. Infection occurs by the transference of the gonococci by the fingers of the mother or the nurse, by clothing, sponges, towels, dirty bath

water, &c.), contaminated by the lochial discharge of the mother or by the infective pus of another infant.

If infection occurs during birth, the first symptoms usually appear on the third day. They consist of slight swelling and reddening of the conjunctiva, and increased secretion as in a case of catarrhal conjunctivitis. The margins of the lids are covered with a dried yellowish secretion; when the lids are separated there exudes a watery fluid of yellowish colour, which sometimes contains grey shreds. As the inflamed conjunctiva bleeds readily, the discharge is sometimes hæmorrhagic in character. The symptoms rapidly get worse. The classical picture of gonorrhœal conjunctivitis is usually fully developed on the second or third day after the appearance of the first symptoms. A creamy pus of more or less thick consistence flows from between the lids, especially when they are separated. The lids are greatly swollen, infiltrated and reddened as seen externally; on exerting them the palpebral conjunctiva is seen to be in a state of severe inflammation; the ocular conjunctiva is often œdematous and injected, and is frequently the seat of hæmorrhages.

The subsequent course is variable; on the one hand it depends upon the violence of the irritant and the resistance of the infant and, on the other hand, it depends upon the management of the case—perhaps this is the more important point. There are, however, cases whose course seems to be quite independent of treatment; some are very severe and fulminating, others are abortive and mild. The great danger of hæmorrhage is the extension of the process to the cornea, which may become infiltrated even before the disease has reached its height. The infiltration affects the superficial and deeper parts of the cornea, and leads to ulceration, perforation, prolapse of the iris and in very bad cases to panophthalmitis. Phlegmonous abscesses in the lids and in the sub-conjunctival tissue are rare local complications.

The termination of the disease depends upon the intensity of the corneal affection. Small infiltrations heal up and leave only very faint streaks on the cornea, which do not interfere with vision. Larger infiltrations result in extensive opacities which are a serious hindrance to vision. Deep ulcers and prolapse of the iris produce more or less thick scars which may or may not include the iris, lead to the development of anterior capsular cataract and to staphylofomous bulging of the cornea. Panophthalmitis will cause destruction of the globe. As the disease is usually bilateral from the start, or if it is at first unilateral the other eye usually undergoes secondary infection, it involves the risk of great loss of vision or permanent complete blindness.

The acute inflammatory symptoms usually last from three to six days, after which the swelling and the discharge decrease gradually. After about three weeks the lids can be freely separated. The total duration of the disease varies from three to eight weeks, but no general statement can be made on this point, because even in neglected cases, the duration of the process is greatly influenced by institution of appropriate treatment.

Generalized gonococcal infection is not often seen in this disease. Arthritis is the complication which has most often been observed (*vide infra*), and possibly this is a process analogous to joint supuration in cases of pyæmia. But even if there be a gonococcal septicæmia (Hochstein), infants do not apparently run any risk therefrom. Secondary infections in other regions such as the mouth, vulva or skin are very rare incidents.

Prophylaxis.

(1) Credé's method, in the words of the author, should be employed in the following manner: "The infant, after the umbilical cord is severed, is washed and the eyes are wiped out with a clean piece of rag soaked in clean fresh water, so as to remove all mucus adhering to the lids. Then the drops are applied, before the infant is dressed. Each eye is gently opened by means of two fingers, and one drop of a 2 per cent. solution of silver nitrate is allowed to fall on the cornea from a little glass rod. No other treatment for the eyes is necessary. If slight redness and swelling of the lids with a secretion of mucus appear within the next 24-36 hours, the drops should not be repeated. The glass rod should be 3 mm. in thickness, with a round well-smoothed end. The nitrate of silver solution must be kept in a dark bottle with a well-ground stopper, and only a small quantity should be prepared" (about 10 c.c.).

The excellent results of this method of prophylaxis is proved by numerous statistics. If the method is properly used, the morbidity is reduced to a minimum, if not a vanishing point. The following figures are given by Dimmer.

| BEFORE CREDÉ | | | | AFTER CREDÉ | | | |
|--------------|-------------------|---|-------------------------|-------------------|---|-------------------------|--|
| | No. of infants | | Cases of Blepharitis | No. of infants | | Cases of Blepharitis | |
| Credé | 2,807 | — | 10.8 per cent. | 1,160 | — | 0.1—0.2 per cent. | |
| Königsstein | 1,007 | — | 4.3 " | 1,210 | — | 0.7 " | |
| Felsenreich | 1,337 | — | 4.3 " | 2,000 | — | 2.9 " | |
| — | — | — | — | 2,000 | — | 1 " | |
| Bayer | 1,106 | — | 22.3 " | 361 | — | 0 " | |
| Haab | 20,000 | — | 9 " | 10,000 | — | 1 " | |

The failures after Credé's method are due to the use of old inert solutions, and to carelessness in the instillation of the drops. It sometimes requires patience to separate the lids of a newborn infant, and it is quite conceivable that in the rush of work at a lying-in hospital it may happen that the drop does not go into the conjunctival sac, but runs over the cheek.

Sometimes the instillation of nitrate of silver causes a conjunctival irritation, which becomes manifest after the lapse of a few hours. This "nitrate of silver catarrh" may be severe, and cause great swelling and secretion, even a purulent discharge. Cramer thinks that the intensity of the reaction does not depend so

much upon the amount and strength of the solution instilled, but rather upon the mechanical damage which the conjunctival mucous membrane suffers during birth, so that it is in a state of hyperæmia and serous infiltration. Nevertheless, the cause of the irritative symptoms which may appear after instillation no doubt partially consists in an improper technique. Bischoff states that the inflammatory reaction very rarely occurs and is always slight, if the method is carried out properly. This "silver nitrate catarrh" usually disappears completely in four or five days, and often before this time.

(2) In order to prevent these symptoms of irritation modifications of *Crédé's method* have been suggested. Leopold uses a 1 per cent. solution, freshly prepared. Hellendal has suggested a method of accurate dosage. The solution is kept in ampullæ of 0.5 c.c.; after opening, the contents are sucked up into an ordinary eye pipette, which is provided with a cotton-wool filter at its lower end. Two drops are slowly expressed into each eye. Willim states that solutions of $\frac{1}{2}$ to $\frac{3}{4}$ per cent. are strong enough for prophylactic purposes.

(3) *Albuminates of silver* may be used instead of nitrate of silver, e.g., protargol in 10 per cent. solution (Veverka) or largin in 5 per cent. solution (Fürst), silver acetate in 1.2 per cent. solution (Thies, Zweifel, Seefelder, Seipades) or sophol, a compound of formaldehyde, nucleinic acid and silver in 5 per cent. solution (v. Herff, Hörder, Gallatin, Hannes, Zemann). The advantage of silver acetate is that it maintains its concentration even after long keeping. Sophol is perhaps the most popular substitute, because of its stability, freedom from danger, painlessness, its non-irritating effect and active bactericidal power (Lefele). Eversbusch advises the use of the chemically indifferent permanganate of calcium (1 in 1,000) in infants whose parents are known to be hæmophilic, owing to the risk of fatal hæmorrhage from the free surface.

A good deal of discussion has arisen in regard to the necessity for this prophylactic instillation as a routine in all cases, or its limitation to cases where gonorrhœa exists, or is suspected. Although the process may be unnecessary in many cases, it is quite harmless and it ought to be universally adopted owing to the great prevalence of gonorrhœa and the extreme tenacity of its virus. Even if it be quite certain that there is no gonorrhœa in the parents, the instillation is useful in that it prevents the onset of a non-gonorrhœal conjunctivitis.

(4) Küstner recommends further prophylactic measures in cases suspected of gonorrhœa. The external genitals of the mother should be irrigated, at the beginning of labour, with a sublimate solution (1 in 4,000), and then scrubbed with a 1 in 1,000 solution for the purpose of disinfection. The vagina should be scraped by means of a soft tooth-brush, and the latter removed by irrigation with a sublimate solution and the use of a brush. He also recommends that the eyes should receive attention before the shoulders are expelled, by removing all material which adheres to the lids with small cotton-

wool swabs which lie prepared in a solution of iodine trichloride (1 in 4,000).

If parents were warned, before and during pregnancy, of the risks which threaten an infant from this cause, it would induce them to seek treatment at the earliest possible moment.

Late infection of newborn infants, by the mother or by other women and children, can be effectively prevented even in lying-in institutions by the adoption of proper hygienic measures. Every infant should have its own toilette articles, and the nurse should wash her hands before touching each child. In this way, all danger of transference of the virus can be avoided. It is important to explain to the mother, that if her finger or diaper, &c., is contaminated with vaginal discharge, the infant may become dangerously infected by contact.

Treatment.

The treatment of gonorrhoeal ophthalmia is productive of the best results. Probably the assertion of Greff, that correct and early treatment prevents all risk of corneal complications, is too sweeping (Schanz, Elschnig), but there is no exaggeration in stating that corneal affections are reduced to a minimum if proper treatment is adopted early. But badly nourished and debilitated infants may fail to respond, even to early treatment.

Silver nitrate is vastly superior to all other silver preparations for treatment. Thorough irrigation of the conjunctiva, once a day, with a 1 per cent. solution, will probably suffice. Most ophthalmologists, however, advise the more energetic procedure of painting the conjunctiva with a 2 per cent. solution. As newborn infants have no eyelashes, the eversion of the lids is somewhat difficult and requires practice. It should be done by pushing the lower lid, in a position of slight entropion, downwards from the lid margin, and drawing from within outwards the index finger of the other hand over the margin of the upper lid, corresponding to the upper border of the tarsus; the upper lid becomes everted by this manoeuvre. The everted lid is fixed with two fingers of one hand, and the conjunctiva with the bulging portion of the conjunctival fornix is thoroughly but not roughly painted with a camel's hair brush or a cotton-wool probe, which has been soaked in the nitrate of silver solution. The excess of silver solution must be washed away with normal salt solution by means of an undine or a swab. The lower lid must be treated in the same way but it is much easier to evert.

The painting with nitrate of silver solution is done once daily, and not more than twice daily. It should be continued for a few days after the cessation of the discharge. Everbush advises that the strength of the solution should be gradually reduced to $\frac{1}{2}$ per cent.

The cleansing of the eyes is also of great importance; when the discharge is profuse, this must be done at half-hourly or hourly

intervals. As soon as any pus has collected in the conjunctival sac it must be washed out. The eye is slightly opened and the cleansing fluid is poured over it, with an outline or out of a spoon or by squeezing it out of a cotton-wool swab. The mere mopping up of the discharge, or wiping it away can be dispensed with, if the cleansing process is in use. The composition of the cleansing fluid is of minor importance. Usually, a wine-red coloured solution of potassium permanganate (1 in 1,000) is employed, which should be made fresh from a stock solution of 2 per cent. strength. The irrigating fluid may also consist of normal salt solution, boracic lotion, or very weak sublimate solution (Hesse). (Gilbert recommends irrigation with ox serum every two or three hours, because it is rich in opsonins which antagonize the gonococci.)

The sensitive skin of the eyelids may have some ointment applied in order to prevent any eczematous changes.

As a rule, compresses should not be used. Compresses of gauze soaked in aluminium acetate may be permitted when there is severe inflammatory swelling of the lids, but otherwise, unless the compresses are applied ice-cold and changed very frequently, the moist heat which they engender may actually promote the suppuration.

If only one eye is affected, the greatest care must be taken to prevent the infection of the other. Occlusive bandages are not very successful with newborn infants. It is better, therefore, to paint the healthy eye once thoroughly with nitrate of silver, or to instil drops of sophol or a similar preparation daily. Watch must be kept for the earliest signs of inflammation, in order, if possible, to nip it in the bud.

The results of this method of treatment are so satisfactory, that it is quite unnecessary to adopt any other measures. Adam recommends in addition to the daily instillation of one drop of silver nitrate solution ($\frac{1}{2}$ to 1 per cent.), the application to the conjunctival sac of Blendliert ointment (5 to 10 per cent.) at intervals of two or three hours. Some approve of this method (Hörder, Wolfberg), others consider it valueless (Bayer). Else employs a 5 per cent. ichthyol vaseline.

Bernheimer recommends aird powder. The eye is thoroughly cleansed and irrigated, the lower lid is everted as much as possible and the powder is applied thereto, with a glass rod, two to four times daily. When the powder has formed an emulsion with the fluid, the everted lid, thickly coated with the aird emulsion is allowed to close up gradually, and at the same time the upper lid is gently raised, thus allowing the entire quantity of aird to penetrate into the conjunctival sac. In the intervals warm wet pads of gauze, soaked in boiled water, are applied. For a few days, a little aird is dusted in the healthy eye, for prophylactic purposes. When the discharge is on the point of cessation, a 1 per cent. solution of nitrate of silver is instilled once or twice daily, for a few days, to prevent relapse.

Everbusch recommends the instillation of $\frac{1}{2}$ to 1 per cent. physostigmin for corneal ulcers situated at the periphery; for those situated centrally $\frac{1}{2}$ per cent. of atropine or $\frac{1}{5}$ per cent. of scopolamin. The first application should be one hour after the painting of the lids, and then at equal intervals, two to three times in the afternoon. When the suppuration has ceased, it is desirable to apply moist warm compresses to the lids. As a rule, treatment by a specialist is urgently required, when the cornea is affected.

OTHER FORMS OF CONJUNCTIVITIS.

All cases of conjunctivitis in newborn infants, even if suppurative, are not necessarily gonorrhoeal. The frequency of non-gonorrhoeal conjunctivitis is indeed very considerable (49 per cent. in Elschig's cases).

Many different micro-organisms have been found, e.g., pneumococci, staphylococci and streptococci, influenza and pseudo-influenza bacilli, diplo-bacilli, Koch-Weeks bacilli and bacillus coli (Dimmer, Credé-Hörder). Elschig found large quantities of the *Bacillus verosis* in three cases. He thinks that this harmless parasite of the conjunctiva finds therein a good nutrient medium if the membrane has been injured by the labour. In some cases no micro-organism has been found at all.

The clinical symptoms of the different forms of non-gonorrhoeal conjunctivitis are not characteristic. The disease may be very mild and subside in a few days, or it may persist for weeks, with the same causal factor. As a rule the course is mild, even if the discharge is purulent (pneumococcal infection); but the discharge is generally serous. It is very rare for the cornea to be affected. The disease usually begins later than the gonorrhoeal type, after the fifth day, but it may be delayed to the second or third week. It is not possible to exclude gonorrhoea with certainty, unless a bacteriological examination is made, and it is best to take the discharge for examination from the conjunctival fornix.

Attention has recently been directed to another form of conjunctivitis in newborn infants, by Hallerstedter and Prowazek, Stangardt, Schmeichler, Lindner, Holschiller and Morax ("Einschlussblenorrhoea"). It is a purulent ophthalmia which occurs early, and the discharge is characterized by containing the same inclusion bodies as are seen in trachoma, and which can be demonstrated by the Giemsa stain. That this is a parasitic condition quite independent of gonorrhoea, is proved by inoculation experiments on the eyes of the apes. The course of this disease is prolonged, but the prognosis is favourable.

The treatment of non-gonorrhoeal ophthalmia is essentially the same as that of the gonorrhoeal variety. In the mild forms it is not necessary to use the nitrate of silver so often, or it may be dispensed with altogether, and treatment be limited to frequent irrigations with potassium permanganate solution, or oxycyanate of mercury 1 in 5,000, &c.

CONGENITAL INFLAMMATION OF THE LACRYMAL SAC.

This condition depends upon the congenital closure of the nasolacrimal duct, previously mentioned, and the congestion in the lacrimal sac resulting therefrom. The pent-up fluid is decomposed by the bacteria which enter from the conjunctival sac, and a secondary inflammation follows. The disease is almost always unilateral. At first the only evidence of it may be a slight formation of pus at the internal canthus, but in severe cases very characteristic swelling and reddening of the lacrimal sac region take place. Pressure in the lacrimal region is followed by the escape of a gelatinous or opaque purulent discharge, or of detritus, from the lacrimal canal.

It is important to recognize this condition, because it may lead to inflammation of the conjunctiva and to infection of the cornea. As spontaneous recovery often occurs, expectant treatment may be adopted at first. Light massage of the lacrimal sac may be performed several times daily to clear the passage; but if this does not lead to the desired result the lacrimal duct must be probed. As a rule, the condition clears up rapidly after the first passage of the probe (Peters, Heissmann, Antonelli, Zenmayer).

(B) Diseases of the Ear.

Our knowledge of ear disease in the newborn is entirely limited to *suppurative inflammation of the tympanic cavity*. Post-mortem examinations suggest that the condition is not at all rare, but it has usually been in badly developed and unhealthy infants that pus has been found in the tympanum.

Urbanowschitsch explains the frequency of purulent otitis media in newborn infants as follows: (1) Interference with involution of the foetal tympanic gelatinous contents; (2) penetration of liquor amnii into the tympanum owing to premature movements of the foetus; (3) possibly also the entrance of the stomach contents during the act of vomiting; (4) the general tendency of the infantile organism to suppurative processes; (5) the great vascularity of the tympanic cavity in infants.

According to Aschoff, liquor amnii may find its way into the tympanic cavity in an early period of foetal life, so that the otitis is not necessarily the result of an infective process, but may be merely due to the presence of a foreign substance. Various bacilli have been found in the pus (*influenza bacillus*, *pus cocci*, *B. Friedländer*, *B. pyogenes*, &c.), but the gonococcus must be especially mentioned, for, according to Haug, it can be found both in the discharge from the ear and the nose. This *oto-bleorrhoea* may run its course, either with or without an accompanying gonorrhoeal rhinitis, and is undoubtedly due to infection during the process of labour.

The clinical signs of otitis in the newborn are not usually very

definite. It is not always possible to be sure that the infant is suffering pain in the ear. It is very difficult indeed for those who are not experts to examine the membrana tympani in infants.

Although most cases of otitis wherein pus is found in the ear at the post-mortem are secondary, the danger of this condition must not be under-estimated. It may be the starting point of a purulent meningitis (Bonhoff and Esch), or of a sinus thrombosis. Most cases of otitis in infants either subside spontaneously or end in perforation of the drum unless the patient succumbs to some other disease. The author has seen a case of bilateral otorrhea in an infant a few days old.

Hæmorrhage may occur from the ear in addition to the purulent discharge in cases of hæmorrhagic sepsis.

CHAPTER IX.

THE SKELETON.

Congenital Abnormalities and Postural Defects.

(1) Trunk.

CONGENITAL kyphosis is remarkably rare. It may affect the entire vertebral column (Lange) or only one segment, e.g., the cervical vertebra (Bernhard). It is probably due to a deficiency of liquor amnii; some instances may be explained by maternal disease.

Congenital scoliosis is perhaps more frequent, and is caused either by the constrained position within the uterus or by the asymmetry of certain vertebrae.

In the thorax there occurs occasionally the deformity which constitutes the well-known funnel-shaped chest, and also the trough-shaped depression of the sternum and adjacent portion of the ribs. It is supposed that the infundibular thorax occurs through abnormal intra-uterine pressure (through the chin, elbow and foot of the child). Occasionally in premature children with thin flexible ribs, a deep depression in the sense of an infundibular thorax occurs with each inspiration.

(2) Extremities.

(A) MALFORMATIONS AND TRANSPOSITIONS.

The following forms of congenital defects of the extremities have been described: Amelia—congenital defect of all the extremities (Rosenhaupt); ectromelia—total absence of one or more extremities; hemimelia—rudimentary formation of the distal part of one or more extremities, with normal development of the proximal part of the extremity; phocomelia—absence of the proximal segment of one or more extremities with presence of the distal parts (fig. 55).

In the last-mentioned malformation hands and feet start straight from the trunk. K. Mayer describes as *symmelia* a formation resembling *phocomelia*, in which the thigh was not absent, but was dislocated at the leg.

These malformations are due, partly to disturbances in development of a degenerative nature, partly to constriction by the umbilical cord, or more frequently to amniotic constrictions and adhesions. Amniotic cords give rise to the so-called intra-uterine amputations, the children being born with stumps of extremities. Finally, the rare transpositions of the larger parts of the body are explained by amniotic bands becoming attached to a certain part of the body (e.g., the foot) during fetal existence, the trunk continuing to grow and changing its earlier position (Ahlfeld). More or less deep grooves may be considered as a preliminary stage



FIG. 55.—*Phocomelia*.

of amniotic constriction, such as may occasionally be found on the extremities, involving the soft parts only (skin, muscle, nerves).

Congenital defects of some long medullated bones cover to some extent those deformities classified as *phocomelia*, such as the rare absence of the humerus and, somewhat more frequently, the femur. With absence of the forearm or leg there are generally simultaneous deformities of the hand or foot, thus: with absence of the radius, the thumb with its metacarpal is lacking; with absence of the tibia, the big toe and first metatarsal are lacking; with defects of the ulna and fibula there is absence of the ulnar and fibular parts of the hands and feet. Whether it is a case of total or partial defect of these bones can only be decided with certainty by means of X-rays (Joachimsthal).

Malformations in the region of the fingers and toes consist in a deficient number (*oligodactylia*), or too large a number (*polydactylia*); duplication of the thumb is relatively frequent, as is also *syndactylia*. To a large extent they are amenable to operative

treatment, either by plastic operations or gradual severance by means of special clamp apparatus (Spitzzy). Supernumerary parts can be removed. This is particularly easy with skin appendages, which are found sometimes symmetrically on the fingers and toes. They are usually supernumerary little fingers or toes, each provided with nails, attached to a thin stump, which can be detached by means of a silk thread.

(B) CONGENITAL CHANGES OF POSITION AND CONTRACTURES OF THE LIMBS.

The most frequent and most important congenital deformity is congenital club foot (*pes varus congenitus*). The foot is fixed in a position of supination, the point of the foot turning inwards and sunken. Club foot is frequently bilateral and sometimes combined with other malformations.

More rare is club hand, i.e., turning of the hand to an angular position against the forearm; it is generally fixed in palmar and ulnar flexion. This affection is often, though not invariably, combined with malformations of the skeleton, of the forearm (defects of bone).

Multiple congenital contractures occur. According to Wunsch, wrist and ankle joints are most frequently affected, the conditions found being the hand in volar, less frequently, in dorsal flexion (Hoffa's *Dackelhand*), club feet, impediment to movement in the region of the hip and shoulder joints, the elbow and knee joints, less frequently in the vertebral column and jaw. The joints may be more or less ankylosed (Keller).

The deformities occur probably most frequently from pressure on the part by the maternal wall; sometimes marks of pressure may be observed on the skin. Amniotic deformities, fetal bone disease and general diseases are considered as aetiological factors.

For multiple contractures Wunsch recommends rectification, tendon transplantation and fixation in plaster-of-Paris as soon as possible.

Concerning the treatment of the most frequent, and therefore, the most important deformity, viz., club foot, opinions of surgeons differ considerably. Some are in favour of preliminary treatment of the foot with rectifying manipulations and actual reduction at the age of nine to twelve months, while other authorities (Finck, Oettingen, Spitzzy, Haude) favour definitive rectification as early as possible, even during the first few days. The most appropriate method of early treatment of club foot is that of Finck-Oettingen; after correction of position, which can be done without tenotomy, a bandage is applied and held firm by some adhesive plaster.

Pes equinus and flat foot should be mentioned among other congenital abnormalities of posture. According to Lengjellner, the foot is flat or sunken in 75 per cent. of newborn infants, and only in

25 per cent. is the arch well defined. Spitzzy states that a certain percentage of infants have feet which tend to the valgus position, but nevertheless only a small percentage have definite flat foot; in these cases the foot is held in the fetal position, i.e., the back of the foot in contact with the leg—just the opposite to club-foot. The treatment is quite analogous to the latter.

A slight inward bending of the tibia is very frequently seen in newborn infants. This is the result of intra-uterine pressure and rectifies itself in the course of growth.

(C) CONGENITAL DISLOCATIONS.

Congenital dislocations of the humerus are extremely rare. Dislocations of the elbow-joint are usually displacements of the radius forwards; the hand is in a position of pronation and cannot be supinated, nor can the forearm be completely flexed at the elbow. If there are also deformities of the radius and ulna, the hindrance to the movement of supination is greater still (Blumenthal). Congenital dislocations of the knee are comparatively rare. They may be associated with absence of the patella. Kuhl states that displacement of the knee-joint in the newborn consists of great hyperextension so that the leg may come in contact with the thigh. In extreme cases, it looks as if the back of the joint were in front. The treatment is to reduce the dislocation, by the bloodless method, and apply a plaster bandage.

The most important of the congenital dislocations is that of the hip. Most of these cases do not become evident until the child begins to walk, and the displacement is not usually very marked in the newborn infant, though the existence of the displacement at birth can be established anatomically. At first, however, it is rather a matter of sub-luxation or of a congenital tendency to dislocation (changes in the acetabulum or head of the femur).

The employment of X-rays renders an early diagnosis possible. Bade points out that there is an alteration in the normal folds of the skin in cases of dislocation of the hip. In normal children the terminal points of the so-called adductor folds (lying in the groove between the adductor and quadriceps femoris) coincide, whether the legs are outstretched or in contact, but in cases of congenital dislocation there is a want of symmetry in these folds.

If the diagnosis has been made, Spitzzy advises not to delay treatment but to employ continuous extension to prevent the development of the displacement.

(D) CONGENITAL (INTRA-UTERINE) FRACTURES.

Fractures *in utero* may arise if the bones are abnormally brittle owing to some foetal disease. Incomplete fractures may also be caused by the constriction of amniotic bands. Direct or indirect injuries to the mother during pregnancy may also cause fractures

of the bone, which may be already united by callus at the time of birth. Such bones are usually much deformed (bent, bowed or shortened). The tibia and fibula are the bones most often broken in this way (Kramer, Rentoul).

(3) Skull

The skull of the newborn infant is subject to frequent and numerous abnormalities, involving the fontanelles, sutures and cranium. Our knowledge of the conditions is due to the exhaustive studies of Kassowitz.

The anterior fontanelle presents the most numerous variations in size and shape. Some fontanelles are not more than half a centimetre in diameter (even as small as 2 mm.), others have a diameter of 3 centimetres. Sometimes the diameter of the fontanelle cannot be measured owing to the width of the sutures which open into it.

The posterior fontanelle is also subject to some variations. It is stated that this is usually closed at birth, but Kassowitz was able to show that it was open in 25 per cent. of a very large number of cases. Its diameter may be more than half a centimetre. R. Meyer refers to the comparative frequency of triangular or square Wormian bones in the region of the posterior fontanelle.

The frontal suture can sometimes be followed to the root of the nose; but it usually ends somewhere between the lower end of the fontanelle and the glabella. Sometimes, however, no trace of a frontal suture can be detected.

In Kassowitz's cases, only 31.7 per cent. presented no abnormalities in the coronal suture. In the others, the sutures were gaping, and the bones which formed their edges were movable or soft. But a gaping suture is not necessarily associated with greater mobility or displaceability of the edges of the bones which are separated by the suture. The softening affects the parietal bones more often than the frontal, whose borders often present a normal resistance in contrast to the flexible parietal bones. Kassowitz also showed that the abnormalities in the coronal suture are very often asymmetrical, and that the right half is more often affected than the left, which corresponds closely to the position of the skull in utero.

The sagittal suture presents more frequent abnormalities than any other cranial suture. These include the shape of the gaps, the displaceability of the bones at the borders and the softness of their edges. The most varied combinations are apt to occur. The abnormalities are so frequent that one may entertain a doubt whether it is correct to assume that a closed suture with hard immovable edges is the normal condition. Kassowitz states that only 12.4 per cent. were quite normal cases.

The lambdoid suture, in contrast to the sagittal suture and also in contrast to its condition in older (rachitic) infants, is normal three times as often as the sagittal, and is also much more frequently normal than the coronal suture. There are not often any gaps in

the suture, nor is softening of its edges at all frequent. The soft places are mostly at the parietal borders, whilst the occipital bone is either not affected at all or only slightly so.

If the softness which is felt at the edges of the bones of the skull extends beyond the sutures to any extent, the condition is known as "soft skull." As already indicated, this softness mostly affects the parietal bones, especially in the vicinity of the sagittal suture and particularly at the level of the vertex. The softening is usually evident on both sides, but it is frequently more pronounced on one side than on the other. The softening feels just like cranio-tables in rachitic infants; in severe cases the bone is replaced by an easily depressed crumpled membrane which extends as far as the parietal eminence. The transition to normal bone is quite gradual.

In addition to this diffuse softening, the nature of which will be discussed in detail later on, there is another but rarer form of defective ossification consisting of numerous gaps in the skull, described by Engstler and Hochsinger as "Lückenschädel." The transition from hard bone to soft membrane is not gradual as in the ordinary "soft" skull; the spaces are sharply defined and consist of tissue absolutely free of bone. Hochsinger calls them pseudo-fontanelles. The gaps are circular, of the average size of a bean, but sometimes larger, and are situated at the level of the parietal eminences or near the sagittal suture, the rest of the skull being ossified all over. Hochsinger remarks that indications of such gaps in the skull exist in the form of uniform indentations in the course of the sagittal and lambdoid sutures. These are situated symmetrically on the suture and unite to form a small circle. The development of these gaps in the skull appears to have some causal association with spina bifida (Recklinghausen, Heubner, Engstler, Hochsinger). It may possibly be an expression of the local arrest of development which occurs in various positions in the cerebro-spinal canal, or it may be the result of intra-uterine arrest of growth consequent upon the pressure exerted in the course of a hydrocephalic process. Wieland observed that the edges of the bone are sometimes bent outwards—an observation which shows that intra-cranial pressure has some etiological significance in connection with the occurrence of these gaps in the skull. The prognosis is good unless spina bifida exists at the same time; the gaps gradually decrease in size quite spontaneously.

The adhesion of the membranes or of the placenta to the foetal head has been known to cause defects in the bones of the foetal skull, but this is an exceedingly rare incident.

Diseases of the Bones.

(A) THE PROBLEM OF CONGENITAL RICKETS.

Although rickets is undoubtedly a constitutional disease, its clinical and anatomical diagnosis depends essentially on changes in

the skeleton. It will therefore be appropriate at this point to discuss briefly the problem of congenital rickets.

This subject has for several years excited much scientific controversy, and the question still remains unsolved. Two principal views dominate the situation. According to the one, there is a congenital predisposition to rickets, though the disease itself is not congenital, but develops in infants with a special diathesis. The damage, which manifests itself in the skeletal changes, occurs during the first few months of life, and the disease itself begins a short time after birth. According to the other view, the disease begins during foetal life, and is caused by the same factors as produce rickets later on. The peculiar conditions found in the skulls of some newborn infants, as already described, are, on this view, to be attributed to rickets. On this assumption the gaping sutures, the widely open fontanelles, the displacability of the cranial bones, the flexibility of their edges are all rachitic changes. The "congenital soft head," apart from the localization, would also appear to be a true example of rachitic craniotabes. In many newborn infants an enlargement of the ribs is to be noted at the junction of the bone and cartilage—at any rate the junctions are palpable. This is regarded as a genuine "rickety rosary."

In discussing the question of congenital rickets one has to decide whether the changes in the skeleton just mentioned are rachitic or whether they are the ordinary results of delayed ossification. Kassowitz and his pupils, Feer, Fischl, Spietschka, and several French authors (Spillman, Marfan, Nau, Porak and Durante) argue in favour of congenital rickets, while the opposite view is taken by Pommer, Tschistowitsch, Escher, Finizio, Fede, and especially Wieland. Attempts have been made to settle the question, both on clinical and anatomical grounds. The extensive researches of Kassowitz show that there are seasonal variations in the frequency of the skeletal changes in the newborn, just as there are in the incidence of infantile rickets, and also that country-born children are better off as regards "rachitic" signs than town-born children even in the early days of life. He concludes therefore that the same poison which causes rickets in infants may already act upon the infant within the uterus. The remarkable localization of the softness in the skull in newborn infants, which is quite different from that of older rachitic infants, is due to the pressure upon the cranium during birth, owing to the downward pull of the brain. Spietschka has traced the subsequent history of infants who were born with soft skulls. He found that the history varied according as treatment by phosphorus was employed or not. Under phosphorus treatment the "craniotabes" was completely cured in a few weeks, and no development of other rachitic changes in the skeleton occurred. The untreated cases showed an advance in the morbid process; the defects already present increased, or the soft sutures developed into an extensive craniotabes in the course of a few weeks. Spietschka only saw one instance of rapid spontaneous recovery of

a congenital soft skull among his numerous cases; as a rule, if spontaneous improvement took place, it was very slow and incomplete.

The opponents of this theory of congenital rickets might explain such conditions thus: that the children with congenital softening of the skull possibly only possess an increased disposition to a rachitic disease without on that account being rachitic. But, in general, it appears that affections of the newly born resembling rickets may, under favourable conditions (breast-feeding, warm period of year) resolve spontaneously without anti-rachitic therapy; otherwise in view of the frequency of softness of the skull many more severe forms of rickets would be seen, for such would be certainly expected with the intra-uterine commencement of the disease. Hochsinger observed a case of softening of the skull which later developed into true craniotabes of the occiput. Marfan reports that the congenital rickety rosary may change into the rachitic rickety rosary of later months. It would be very desirable for there to be a larger number of cases available which would enable one to decide whether the congenital symptoms resembling rickets are precursors of a genuine infantile rickets. Regarding this question, Kassowitz himself had no personal experience, but, judging by Wieland's cases, he believes that the softness of the skull bones may quickly improve under the influence of a favourable time of year, though the soft skull bones of children born in the winter time show no tendency to improvement.

Concerning the histological changes, Wieland, on the basis of thorough examinations, comes to the conclusion that at the plates with endochondral ossification, neither the deposit of lime in the degenerated area, nor the distribution of vessels in the cartilage, nor the condition of the various zones of hypertrophy of cartilage show such changes in the newly born, that they might be considered incipient or advanced rickets. The interpretation of the conditions of the skull bones as rachitic is derived, in Wieland's opinion, from confounding the "physiological osteoid" of newly born with the morbidly increased rachitic osteoid. In the rapidly growing bones of the normal newly born and premature children much bone tissue is found devoid of lime salts. The thickness of this physiological osteoid is, in contrast with rachitic, almost constant. A measurable widening of the osteoid sutures, the solitary entirely characteristic histological stigma of the rachitic process, is not found before or during birth. Kassowitz with the aid of more recent research, has sharply criticized this interpretation of the histological aspect. He does not acknowledge the occurrence of a physiological osteoid, as he has never been able to prove it macroscopically in normal bones. According to his opinion, histological researches prove the rachitic nature of congenital rickety rosary, and also the identity of congenital softening of the skull with rachitic craniotabes.

As long as the various authorities disagree what, under the microscope, may and may not be regarded as rachitic, the question

of congenital rachitis must remain undecided. In spite of well-founded theoretical objections the wisest standpoint for the practitioner is to admit the possibility of an intra-uterine existence of rickets, for the prophylactic and hygienic measures, recommended by Kassowitz, are certainly advantageous to mother and child; the pregnant mother should be as much as possible out of doors, and in the colder months her rooms should be thoroughly aired. In respect of changes of skeleton in the newly born judgment should at present be withheld. If they are stationary, or if unmistakable rachitic symptoms occur, phosphorus cod-liver oil is highly to be recommended.

(B) FETAL DISEASES OF SKELETON.

The so-called fetal diseases of the skeleton possess a rich terminology. In the earlier literature there is a whole string of names, but for which there are essentially, as Kaufmann's researches have shown, merely two varieties of disease forming a basis of classification, viz., chondrodystrophia and osteogenesis imperfecta. Both types used to be included under the name "fetal rickets." Though the question of congenital rickets has not yet been solved, there is at least unanimity on one point, viz., that the above-mentioned diseases of the skeleton have no connection with rickets.

(a) Chondrodystrophia fetalis.

MICROMELIA.

The name micromelia signifies the essentially clinical symptom of the disease, shortness of limb. Nowadays it applies only to chondrodystrophia, whereas the term phocomelia (also shortness of the extremities) and certain forms of osteogenesis imperfecta are not included in the term micromelia. Among other terms used nowadays osteosclerosis cong. and achondroplasia may be mentioned. The terms micromelia and chondrodystrophia are not entirely synonymous, as, according to Kaufmann, there is a special form of dystrophy not associated with shortness of the extremities, viz., chondrodystrophia hyperplastica. The usual form of chondrodystrophia, ch. hyperplastica, and ch. malacica, representing the most extreme degree, are clinically expressed by the remarkable shortness and clumsiness of the long bones, and a remarkable disproportion between the trunk and length of extremities (figs. 36 and 37). In the thickening of the extremities the skin also participates, which is frequently exhibited by increase of subcutaneous fat and deep transverse furrows. The fingers are also short and thick. The head is relatively large, sometimes abnormally so, well ossified, the anterior fontanelle is wide open, the root of nose generally much drawn in, the lips thick, the mouth open, and tongue protruding. According to Bohrenträger, the type of face resembles "one about to sneeze" (Sumita). The thorax is

described as bell-shaped. The abdomen is, as a rule, distended, and the vertebral column very lordotic in the lumbar region.

The changes in the skeleton are derived essentially from retardation of the epiphyseal growth of bone, with normal periosteal growth. The structure of bone produced under the periosteum is very dense; it results in the coarse sclerotic quality of bone (fig. 56). The formation of synostoses takes place in the synchondroses and syndesmoses; the junctions of the epiphyses close prematurely, the further growth in length is impeded by coalescence of the latter. The endochondral ossification ceases prematurely owing to dystrophy of the cartilage, and deficient or abnormal hypertrophy of cartilage.

Whereas Kaufmann and Sumita consider chondrodystrophia a malformation, in fact as a vitium primæ formationis of the cartilage, Abels considers it to be due to a disturbance in the internal secretory equilibrium. He points out the insignificant but frequent changes



FIG. 56.
Chondrodystrophia. (Observation by E. Meyerhofer.)



FIG. 57.

Chondrodystrophia. (Observation by E. Meyerhofer.)

in the thyroid gland and the abnormal development of the genitals which have been observed in some cases; also the central nervous, muscular and digestive systems (liver) show, in his opinion, overdevelopment, which indicates an abnormally increased productive stimulus. Abels does not regard the condition as due to subfunction of the thyroid gland and thyrodysplasia (in the sense of Moro), but to hyperthyroidism. In respect of this he refers to a case reported by Cavazzani; before and during her pregnancy a woman had taken considerable quantities of thyroidin tablets, and the product of this pregnancy was a typical micromelia.

A large number of cases of chondrodystrophia are born dead or die immediately after birth. Relatively frequent premature birth occurs. In milder forms of the affection the children can survive the first few days, and in this case the prognosis is more favourable. According to Kassowitz it appears "that the affection only threatens

life during the fetal period, but that when this critical period is past the viability is not seriously imperilled by the malformation." There are many cases on record of micromelous infants and older children, so that one can obtain from the literature an unbroken series from the intra-uterine period to a ripe age (Sumita). Micromelics who survive develop into the so-called *chondrodystrophic dwarfs*, individuals who, in respect of intelligence and sexual functions, show no deviation from the normal.

According to Abels the relation of the circumference of the head to the length of the body gives a certain clue to the severity of the



FIG. 48.—Skilograph of legs, fig. 52.

affection, and the viability of the child. In normal newborn children it is about 2:3, the circumference therefore of the head being about 67 per cent. of the length of the body. In *chondrodystrophia* this quotient may be more than 120 per cent. If the disease is not so extreme as to endanger intra-uterine life, yet the shock of birth and the sudden change of conditions of existence may result in death. Of children that survived only a short time, none has exhibited a higher measure than 100 per cent. (Stöltzner, length of life 1 hr., 92.5 per cent.; Sumita, length of life 4 days, 92 per cent.). The viability appears to start with a proportional value of about 80 per cent. in typical micromelia.

(2) Osteogenesis imperfecta (Vrolik)**OSTEOPATHYROSIS FETALIS (HOCHSINGER).**

The most characteristic symptom of the disease lies in the abnormal brittleness of the bones. The calvaria are remarkably soft and thin (Hochsinger's thin skull), and exhibit after birth multiple incomplete fractures (Harbitz). The long bones of the extremities, the collar-bone, and ribs are often the seats of numerous and incomplete fractures owing to abnormal thinness of the cortical layers, hence the extreme deformities, curvatures, flexures and shortenings.



FIG. 59.—Osteogenesis imperfecta.
(Observation by E. Hapfelolder.)

The extremities may literally be bent into a circle (fig. 59). Otherwise the condition of the child presents nothing abnormal. The facial expression is normal, the nose is well formed, and the skin not remarkably thick.

The essential changes in the skeleton consist of disturbance of the periosteal and endosteal formation of bone with undisturbed, almost normal course of the enchondral processes of ossification (Sumita). As the callus calcifies fairly rapidly in osteogenesis imperfecta, annular swellings may develop on the long bones and ribs (hence the old term *militia facialis annularis*), and the fractured parts of the body assume a deformed appearance. The shortening of

the extremities occurring from multiple fractures is always only apparent, and the similarity with chondrodystrophic micromelia only external. On examination by X-rays the characteristic anatomical peculiarities of osteogenesis imperfecta, the multiple fractures, the deficient periosteal disposition of bone, and the extraordinary thinness of the cortical layer, &c., may be easily established (fig. 60).

Concerning the aetiology of osteogenesis imperfecta, observations hitherto have offered but slight clues. Sumita sums up as follows: Thyrogenous causes, direct maternal influences, inflammatory processes, trophic disturbances, &c., may be disregarded; on the other hand, the evidence for assumption of a malformation is relatively sufficient. Hochsinger considers the disease as the result of panostitis.

Most children with osteogenesis imperfecta die shortly after birth or are born dead, but numerous cases have been described in which the children live for weeks and months (Hecker, Stilling, Schreib-Esser, S. Müller, Buday, Hohlfeld, Nathan, &c.).

The disease of later life, described as osteopathsia idiopathica, which is characterized by abnormal brittleness of bone, must also be attributed to a congenital disposition, and is essentially the

same disease as *osteogenesis imperfecta* of the newly born. Looser speaks of the two as *osteoposathyrosis congenita* and *tarda*.

The most important clinical and anatomical differential diagnostic features of the two diseases are set forth in the following table by Sumita:—



FIG. 50.—Skilograph of fig. 59.

Chondrodystrophia.

- (1) Considerable micromelia.
- (2) Well ossified, large head.
- (3) Frequently premature synostosis.
- (4) Frequently retraction of root of nose.
- (5) Quadrangular or triangular hands.
- (6) Firm, massive diaphyses.
- (7) Jagged line of ossification.

Osteogenesis imperfecta.

- Insignificant micromelia.
 Soft head, small in circumference.
 Absence of synostosis.
 Absence of retraction of root of nose.
 Fine, delicate hands.
 Weak, brittle diaphyses.
 Perfectly regular line of ossification.

*Chondrodystrophia.**Osteogenesis imperfecta.*

- | | |
|---|--|
| (8) Presence of periosteal new bone. | Absence of periosteal new bone. |
| (9) Disturbed enchondral ossification. | Normal enchondral ossification. |
| (10) Undisturbed periosteal and endosteal ossification. | Disturbed periosteal and endosteal ossification. |
| (11) Almost invariably normal clavicle. | Clavicle affected. |
| (12) Frequent combination with malformations. | Combination with other malformations rare. |

(C) ACUTE DISEASES OF BONES AND JOINTS.

Acute inflammatory diseases of the osseous system are very rare in the earliest period of infancy. Elgart reports on some cases of osteomyelitis. He cites a case of congenital osteomyelitis of both femora (Senn), and records an analogous case of a child that appeared to be healthy after birth, and fell ill on its tenth day with fever and swelling of both thighs; incision, recovery; the mother had suffered for four weeks before delivery from typical poly-arthritis. Eckstein reports on the occurrence of osteomyelitis at the lower femoral epiphysis during the second week; the disease was perhaps in connection with an umbilical affection; the pus removed by incision contained *Streptococcus mucosus* in pure culture.

Bitner and Zarff report on the occurrence of isolated, acute, purulent coxitis at an early period of infancy. A case of Zarff's manifested even on the twelfth day fully developed clinical symptoms. These consist of painful swelling of the hip-joint (the right one in the five cases on record) and its vicinity; most of them were accompanied by fever; the leg was fixed in the pathognomonic position for coxitis. The disease may apparently last for weeks. During this period the pus erodes the articular cavity and head of femur, and finally breaks through at the back, forming a periarticular abscess. The process may spread by continuity or metastatically (erysipelas, peritonitis, meningitis), or lead to severe general sepsis, which finally kills the child. In one case Zarff observed spontaneous recovery after pronounced symptoms of coxitis. According to Bitner, it is only possible to save the child by operation (resection of the head of femur and free opening of the joint). In all cases hitherto examined streptococci and staphylococci were found. The portal of entry could not be determined with certainty. The infection probably takes its origin from the navel or external skin (intertrigo).

An equally rare, but possibly the relatively most frequent, form of arthritis which has come under observation during the newly born period is arthritis gonorrhoeica. It has frequently been observed as a complication of conjunctival blennorrhoea (Deutschmann, Weiss and Klingenhöfer, Clément, Neuburger, Hechsen,

Brehmer, Paulsen, Hawthorne, Nobécourt and Vitry, Nunn, Goto, &c). As a rule, it does not appear before the end of the second week. It may attack various joints: shoulder, elbow, hand, jaw, hip, knee and ankle. The diagnosis may be established by exploratory puncture; Goto found gonococci in the bloodstained muco-purulent fluid in one case. But, as a rule, punctures for diagnostic purposes should not be done at the onset at least. Gonorrheal arthritis generally offers a favourable prognosis; with ordinary antiphlogistic treatment complete recovery occurs after three to five weeks without disturbance of function. In fatal cases there is probably a mixed infection.

The common form of pyemic arthritis with multiple suppuration of the joints generally occurs during a later period of infancy. In such cases one finds a distinct swelling of a cushion-like nature with deep fluctuation at several joints, particularly at the wrist and shoulder joints; the skin is tense and shiny, but seldom red. The prognosis is unfavourable.

There are some cases on record of "rheumatic swellings of joints" in children a few weeks old (Marshall, Besanquet, Japha). But it is questionable whether they are really examples of acute articular rheumatism (Schlossmann).

SUPPLEMENT.

Congenital Tumours of the Sacro-coccygeal Region.

SACRAL TUMOURS.

Congenital tumours of the posterior part of the lower portion of the body are termed sacral tumours. Stolper divides these into tumours that arise through disturbances in the union of the embryonal component parts of the posterior end of the trunk and tumours representing double embryonic rudiments. The first group comprises dermoid cysts and fistulae, also sacculations of the contents of the spine, including spina bifida occulta; the second group, complete and incomplete double formations and the so-called embryoid teratomata. The term "sacral tumour" generally denotes dermoids and teratomata of various histological structure and size, and situated on the dorsal surface. The size of the tumours varies considerably. As a rule they are very large, sometimes larger than the head of a child (fig. 61). The foetus is frequently born prematurely; not uncommonly it dies before or during birth. The dimensions of the tumour may be so enormous that it creates an obstruction to birth, rendering it necessary to dismember the child (R. Keller). But, as a rule, the cystic tumour is so easy to compress that birth occurs spontaneously (Eirke). If the children survive the tumours increase rapidly in size after birth. The thin skin ulcerates and becomes infected.

Speedy excision is therefore indicated; it has often been undertaken with good results. Bartel's observation of a small cystic congenital epidermoid in an adult shows that these tumours may permit of a long life.

Corresponding tumours on the ventral surface of the scrotum are, as a rule, latent at first.



FIG. 61.—Squid tumour.

CHAPTER X.

SKIN.

I. Congenital Changes of Skin.

Congenital Skin Defects and Scars.

So-called congenital skin defects are rare, although many have already been observed and described (Hofmann, Dittich, Vörner, Lirdig, Keller, Schulte, Sitzenfrey, Kehr, &c.). They are nearly always situated on the head, chiefly at the vertex; their size varies considerably, from the size of a pin's head to that of a five-shilling piece; they are generally circular in form, more rarely longitudinal or irregular, the edge is generally sharply defined as if it had been punched out (fig. 62).

As a rule the defects involve the skin only, or the subcutaneous

connective tissue as well. In rare cases they may penetrate deeper, as far as the pericranium or the bony vault of cranium, exceptionally right through the bone as far as the cerebral surface (Kehrer).

Immediately after birth the defects appear in the form of ulcers under the level of the skin, the base of which is only seldom a fresh wound surface, but is generally covered with brownish scabs, old coagulations of blood. In other cases scars have already formed. According to the stage of the wound the parts surrounding the defects vary: sometimes there is a tumid red rim, sometimes a pale cicatricial or tough border.



FIG. 62.—Congenital skin defect at the vertex of a child 3 days old.

Concerning the origin of congenital skin defects, the general opinion of authorities is that adhesions of the ectoderm occur with the amnion owing to disturbances of development or inflammatory processes; at the places of adhesion the formation of squamous epithelium does not occur. With increase of liquor amnii the adhesions are drawn out into the so-called Simonart's ligaments. If these are torn away from the cutaneous surface of the fetus circular skin defects arise. The accuracy of the theory that amniotic adhesions are responsible for the existence of congenital skin defects could be proved histologically by Sitzentrey. It has already

been shown by clinical findings, viz., the relatively frequent occurrence of amniotic constrictions of the limbs and congenital clefts (A. Deutsch).

Congenital skin defects are sometimes isolated, sometimes in groups. In rare cases they are also found in other parts of the body besides the vertex (e.g., Hoefel's case, on the back). In Deutsch's case, apart from typical defects of the skull, there were plaques at various parts of the body of extremely thin skin, giving the appearance of blisters, and identical with the changes of skin described by Oppenheim in adults as *anetoderma maculosa* (*Dermatitis atrophicans maculosa*).

The prognosis of typical congenital skin defects is so far unfavourable, for most places devoid of hair remain so. It is really a case of *aplasia cutis congenita*, a total absence of epithelial tissue (the name *atrophia cutis congenita* does not correspond to the facts); if the rudiments are lacking there is naturally no subsequent growth of hair.

In the differential diagnoses instrumental (criminal) injuries only come into consideration, so that the affection is primarily of interest in forensic medicine. The absence of any reaction in the neighbourhood of the defect, the frequently circular shape of the tumour, and, as a rule, the normal course of birth, are against a diagnosis of injury.

Swoboda points out that congenital scars may come from angioma, &c., healed during intra-uterine existence.

Retention of Secretion in the Sebaceous and Sweat Glands (Milia, Miliaria).

In a large number of newborn children, both premature and those carried to full time, there are whitish or yellowish puncta the size of fine sand close together at the tip of the nose and alae nasi, and situated just on or above the level of the skin (fig. 153). If the area involved is at all extensive, similar, but only stray, puncta may also be found on the cheeks, forehead, lips, and even on the ears. They arise from retention of secretion in the sebaceous glands which is not caused through closure of the mouth of hair follicle, but through physiological hypersecretion during the last few months of pregnancy. The contents collect in the excretory duct. If one of these puncta is pricked a thickish liquid substance is evacuated (fat, cholesterol, epithelial debris). The milia completely disappear after a while, either in a few days or weeks.

Only very exceptionally do these sebaceous cysts spread all over the body. Hinselmann observed a case of widespread "miliaria sebacea" lasting until the second week, and combined with inflammatory symptoms like *acne*.¹

¹ ABHANDLUNG—A. Kewin (Leck). *J. Dermat. u. Syph.*, 116, 1914, 724) proposes to describe the formation known as "milia" as "comedones retentivum" and the subsequent inflammatory affection as "acne retentivum."

From these sebaceous affections must be distinguished similar congestive phenomena in the sweat glands. In the above-mentioned case Hinselmann found in the skin of the sole congenital sweat gland cysts (congenital hydrocystoma), shiny vesicles resembling cooked sago, with watery contents. A less rare, though uncommon, localization of miliaria in the newly born is the face, forehead and



FIG. 63.—Miliaria (forehead); milia (nose and cheeks). Child 4 days old. (After Leiner.)

neighbouring scalp skin. Leiner describes the condition found as closely collected vesicles resembling dewdrops, which lend a peculiar grey colour to the otherwise unchanged skin; the skin feels uneven and granular to the touch, the vesicles rupture easily, and the fingers are covered with moisture (fig. 63). Leiner attributes the existence of this miliaria to sucking, as it disappears some time after

the feed, and reappears again with the next feed. The vesicular eruption may remain for days unchanged without remission; in rare cases a slightly inflammatory areola forms round one or more vesicles. Miliaria occurs independently of external temperature. Besides hypersecretion of the sweat glands occlusion of the excretory duct might also co-exist, possibly caused by the layer of vernix caseosa.

Treatment of the above-mentioned changes of the skin is superficial; if they are very marked, simple ablutions with soap and application of powder are recommended.

Telangiectases, Angiomata, Nævi.

The many congenital changes in the skin belonging to this group will not be discussed in detail. There is just one question always put to the medical attendant immediately after the birth of the child, viz., concerning the prognosis and the necessity of treatment. It is quite comprehensible that the parents would like to receive a definite forecast regarding the disfiguring vascular nævi and pigment spots on the face, whether they will disappear, or remain stationary or increase in growth.

In many newborn children more or less intense red spots are found in the vicinity of the forehead, mostly in the form of a streak, about the breadth of the little finger, sometimes straight, sometimes irregular, but always sharply defined, which extends upwards, straight or irregularly, from the root of the nose to the hair border. Similar spots and streaks are often found in the region of the eyebrows. If one just strokes over the affected part of skin with the finger the redness immediately disappears and as quickly reappears; there is, therefore, an enlargement of the cutaneous vessels. Otherwise the skin is quite normal. These spots and streaks, commonly known as "stock bites," tend to disappear completely in the course of weeks or months; therefore the cosmetic worries on the part of the parents may be tranquillized with an easy conscience.

But there exists a close relationship between these changes of skin and nævi vasculares, which, on an otherwise perfectly normal skin surface, appear as extensive maculæ and sometimes cover large surfaces of skin, being generally irregular and bright red. The face is a favourite locality for these nævi, although they also occur in other parts of the body. They may become less post partum, but the prognosis is generally by no means so favourable; they may not only remain stationary, but in the course of time increase in size. As a rule, nævi vasculares are darker than the aforementioned harmless telangiectases, in fact, blue red.

The diagnosis of serious changes in the future is certain if one is dealing with a case of nævi vasculares with an irregular, rough, swollen surface, and tumour formations which lead to true angioma. In the latter, of course, there is no hope of spontaneous disappearance; one can only be thankful if such skin tumours do not

increase in size. The treatment (destruction by cauterization, extirpation), which is a simple task with small naevi, should best be undertaken during infancy, though not in the newly born period. As they are benign tumours, only cosmetic considerations are concerned.

The same remarks apply in regard to the prognosis and treatment of the various forms of pigmented naevi and allied skin affections.

Reference should be made here to that variety of pigmentation which has been termed "Mongolian spots," owing to the fact it is almost physiological among Japanese and Chinese. Cheinin found these spots in 80 per cent. of the children examined under a year old. These Mongolian spots are only met with very rarely among European infants (Epstein, Adachi and Fujisawa, Menabuoni, v. Koos, &c.). Epstein of Prague found one among 600 cases, and Koos of Budapest one in 500 cases. According to Koos these pigmented spots only occur in subjects whose general pigmentation is intense; he shows that the condition is not peculiar to Japanese and Chinese, but that it occurs among Esquimaux, Malays, Siamese, the natives of the Philippines and Samoa, South American Indians and Negroes. The few European cases have occurred in Bulgarians, Italians, Germans, Bohemians, Russians and Dutch. In Austria the condition principally affects children of Magyar origin (Spork, Zarff).

According to Epstein, the spots are very much like pigmented naevi; but they are distinguished from the usual brown or dark-brown pigmentation by a distinctly less intense coloration and by their characteristic situation on the sacral or gluteal region. The spots very rarely extend from this region or appear in other places, such as the trunk, the limbs or face (Zappert). The size of the spots varies from a shilling to half a crown, but they may be smaller or much larger; sometimes a number of small spots are found close to a large one. They are usually circular in form, more rarely oval, band shaped or irregular; their border is rather sharply defined. Their colour is of a delicate grayish blue, and slightly suggests a subcutaneous hæmatoma. The spots tend to disappear in the course of a few years and eventually vanish completely. Even among Mongols it is very exceptional to find the spots after the fifth to the eighth year.

Epstein points out that these spots affect the sacral region in common with various other pathological disorders and abnormalities, viz., grooves and furrows, naevi, hypertrichosis, cutaneous poils and cutaneous appendages, &c. This region is subject to these abnormalities because it is differentiated late in foetal life, and it is for the same reason that there is often a very considerable accumulation of pigment in this part.

Congenital Tumours of the Skin.

A few cases of *congenital sarcomatosis of the skin* are to be found in the literature (Odstrčil); three are described by Ahlfeld,

One was a congenital spindle-celled sarcoma of the left leg, the second was a similar case on the right leg, and the third was a congenital pedunculated spindle-celled sarcoma of the right shoulder region. In all these three cases the primary focus is probably to be ascribed to the skin. Randolph has described a case of tumours scattered irregularly over the skin, which was explained as a multiple metastasis of an angio-sarcoma of the kidneys. The only definite case of primary congenital sarcoma of the skin is that of Krause, which was a spindle-celled sarcoma of the size of a pigeon's egg, and was situated below the umbilicus. It was removed and the infant recovered.

Solitary fibromata of the skin are usually congenital, and the multiple fibromata are frequently so (Jarisch). Recklinghausen's disease, which is a neuro-fibromatosis, has been observed in young infants (E. Hirsch).

Congenital Ichthyosis (fœtal or intrauterine)

CONGENITAL UNIVERSAL HYPERKERATOSIS.

Congenital ichthyosis is a somewhat rare disease. Riecke collected fifty-four cases from the literature in 1900; since then there have been several more recent cases (J. Neumann, Pinizio, Haus, Humbert, Ehrmann, Moore and Warfield, Zumbusch, Huebschmann).

The cutaneous surface of infants affected by typical congenital ichthyosis is made up of firm, horny, smooth, yellowish white or greyish scales or plaques which may be over $\frac{1}{2}$ cm. in thickness. They are of varied shapes, circular, oval, triangular, square or rectangular; they also vary in size. Numerous furrows of varying width and depth run between the scales. These furrows may be provided with a thin whitish investment of epithelium, or they may extend as far as the corium. As a rule, the entire skin is affected, with the exception of the hands and feet. The eyes appear to be replaced by red swellings, which are the eyelids in a state of retraction. The lips are also extruded; the open mouth looks like a fish's mouth surrounded by a huge swelling of mucous membrane. The nose is much flattened, and scarcely projects beyond the level of the adjacent skin of the face; the nostrils may be blocked up by horny material, or they may gape. In the position of the auricular cartilage there are flat nodular structures with transverse slits, and the aperture of the external auditory meatus is usually extremely narrow (fig. 64).

These infants are usually stillborn, or if alive may survive for an average period of three weeks, the cause of death being the abeyance of the function of the skin or general infection. The birth is often premature.

Riecke distinguishes a second and rarer type of this disease as *Ichthyosis congenita levata*, the symptoms of which are milder and are only partial in their appearance; moreover, the infants may live

longer. He also distinguishes a third type as *Ichthyosis congenita tarda*, wherein symptoms may be very slight or entirely absent at



FIG. 64.—Congenital ichthyosis.

birth, but they develop quite definitely after a period of days, weeks, or months. The relation between congenital ichthyosis and the

ordinary ichthyosis of later life is still a subject of discussion. They differ in their course and prognosis, but the histological appearances are in many respects very similar, so that Huebschmann does not consider that the two diseases are as essentially different as many other observers hold.

The disease probably begins in the last months of pregnancy. It consists of an overgrowth and heaping-up of the keratin cells, being an abnormality of keratinization of the skin which apparently depends upon a defective germinal layer. This view is supported by the fact that the cases are hereditary and run in families. It is still to-day not possible to decide how much importance should be attached to the internal secretory organs and their relationship to ichthyosis congenita. Moore and Warfield believe that the origin of the disease is to be found in deficient thyroid function, and therefore trophic disorders may be of significance. The same authorities also found pronounced changes in the thymus gland, among these an increase of Hassall's corpuscles; Huebschmann has found similar conditions; Haus believes in a connection between abnormal development of the central nerve system and the skin; he refers to the genealogical table of a family dating back to the year 1688, in which many cases of ichthyosis congenita and mental disease have occurred. Considering the hopelessness of the prognosis, it is superfluous to speak of treatment. In the milder forms, baths and indifferent ointments are best administered.

Ichthyosis Sebacea.

SEBORRHEA SCUMOSA NEONATORUM.

The disease known as ichthyosis sebacea must in no way be confused with ichthyosis congenita. Whereas in the latter case it is a very serious disease, in the former one can hardly designate it as a disease, but only a variety of a normal condition of skin. Some children do not have that fineness and softness of skin, peculiar to the newly born, but a remarkably dry, coarse, cracked and sometimes brownish epidermis. Their skin gives the impression that it is too large for the child. As Leiner puts it, in these cases the elasticity of the skin is reduced, and with every movement, especially crying, numerous tiny folds are formed which remain for a long time, and result in almost invisible cracks. The hands and feet often possess the quality of leather; the folds are then bigger, the rhagades more distinct and owing to their wide raised margins they stand out from the surrounding part. In the depths of the fissures a glimpse of the reddened epidermis is seen. If, during the course of the following few days the cracks and fissures in the skin branch forth over the surface, the epidermis seems to be covered with a network of fine and coarse fissures which enclose large plaques and layers of epidermis (*cutis testacea*). The cause of this remarkable condition of skin is supposed to lie in increased seborrhoea combined with rapid drying of the sebum. Török has therefore proposed the term "*Seborrhoea squamosa*."

The children who manifest this quality of skin after birth generally exhibit at the end of the first week a particularly extensive desquamation (*Superdesquamatio membranacea*, Braun; *Desquamatio lamellosa neonatorum*, Terak) (fig. 65). From these transverse fissures of the epidermis large flakes which feel greasy may often be torn off the body. The desquamation may last for days, in extreme cases even weeks. The affection has as little to do with true ichthyosis congenita as with ichthyosis vulgaris, erythrodermia desquamativa, or dermatitis exfoliativa.

The treatment consists of baths (bran baths) and application of indifferent ointments or sulphur ointment.



FIG. 65.—*Desquamatio lamellosa*. (Child 8 days old.)

The name "*Vernix caseosa pellicularis*" (Bar, Hinselmann) designates a peculiar form of flake-like desquamation which has sometimes been observed after birth. The children look wasted; the body is covered with a thick covering of epithelia and lanugo hairs, which falls off in thick flakes from the skin, which is otherwise normal and free from inflammatory symptoms. The cause of the phenomenon is probably a close adhesion of the epithelium in the superficial layers of epidermis, which prevents it from releasing isolated cells or groups of cells as in the ordinary formation of vernix caseosa; separation of layers from the underlying skin forming a connecting layer only follows from birth trauma.

(II) Exanthemata.

Physiological Erythema Neonatorum.

Our thanks are due to the observations of Leiner whose clinical differentiation of the various forms of erythema in infants, ranks so-called erythema neonatorum as a physiological phenomenon. Nearly every newborn child, as soon as it is freed, during the first bath, from vernix caseosa, and the skin circulation set going,

and the temperature rises after initial depression, manifests a more or less pronounced hyperæmia of the skin. The greatest individual variety pervades. The erythema may exhibit all degrees, from a delicate pink to an intense redness—but a certain degree of erythema must be a criterion as to health. It is obviously a physiological reaction of the cutaneous vessels to the stimulus of the



FIG. 66.—Erythema toxicum neonatorum. Child 4 days old. (After Lehar.)

cooler external temperature, by which the still incomplete function of the mechanism regulating the temperature is revealed. Premature children often manifest the erythema to an extreme degree. The quality of the skin is of essential influence on its intensity. Children with delicate, velvety skin generally manifest relatively

intense erythema, whereas, in children possessing coarse, dry skin (ichthyosis sebacea) it is usually slight or almost altogether absent.

The erythema generally reaches its height at the end of the first or second day and then gradually fades away. Then follows a state of desquamation, which must be regarded as the result of the



FIG. 47.—Erythema toxicum neonatorum. Child 6 days old. (After Lainez.)

erythema and also as a physiological manifestation. The exfoliation is slight and branny. The pronounced lamellar desquamation, observed in some children, is less connected with erythema than with the before-mentioned ichthyotic condition of skin.

Erythema Toxicum Neonatorum.

Leiner gives this name to a transient eruption which often appears in children during the first week and is probably well known to most medical men with any experience in this particular sphere. He differentiates two types of dermatosis, not unlike each other. The first type corresponds to simple erythema and is characterized by the appearance of efflorescences, varying from the size of a pin's head to that of a shilling, seldom rising above the level of the skin and giving a sensation of slight infiltration when lightly stroked with the finger. The lesions are closely set, and sometimes coalesce so as to form large patches or become connected with one another by delicate linear offshoots. Their colour is intensely red or vermillion, making them easily distinguishable from the physiological redness of the skin of the newly born. The erythema is spread all over the body; no favourite locality exists, as it occurs with greater intensity sometimes in one part, sometimes in another. Flexor and extensor surfaces are equally attacked. If the eruption is most marked on the face, a frequent occurrence, one is reminded for the moment of the rash of measles, especially if the eyelids are slightly puffy. From the face the erythema spreads to the scalp, which is often completely covered with it. The palms of the hands and soles of the feet are generally spared or manifest only isolated lesions.

Simultaneously with the erythematous macules sometimes small papular efflorescences occur, which are, as a rule, situated more centrally in the erythematous area and are distinguished from the red peripheral area by their whitish colour. They are raised above the general surface of skin, and by their colour and their flattened surface remind one of small wheals. On drawing the finger over this area of erythema, the red colour of the external part disappears for a few seconds, and there only remain the protruding flat pimples of a white porcelain or—with associated icterus—a yellow colour. The papules are of a fairly dense consistency. Through confluence of the erythematous macules, a formation of large areas occurs which are covered with thickly aggregated urticarial wheals. They are not infrequently found on the posterior aspect of the body, especially in the gluteal region. Both these types may exist simultaneously in the same individual, or may occur singly; almost invariably transitions from one form to the other are found (figs. 66 and 67).

The erythema occurs regardless of the constitution of the newly born child. Both in healthy and sickly children it represents a harmless disease and pursues its course without any particular complications, without much irritation and appears to have no disturbing influence on the further development.

The course of the erythema is short, seldom beyond forty-eight hours, but occasionally, after complete disappearance, fresh eruptions arise. Disappearance does not occur simultaneously over the whole

body, but gradually, so that isolated parts (face, back) still exhibit the erythema, whereas other parts of the body resume a normal condition. The erythema leaves behind no residues, no pigment spots, no desquamation. During the existence of the erythema and for a long time afterwards, the skin is in a state of increased irritability, made obvious by distinct dermographism. Treatment of this harmless eruption is superfluous; light powdering is sufficient.

It is Leiner's opinion that these changes in the skin are connected with dyspeptic intestinal disorders—hence the name *erythema toxicum*. Owing to the frequency of symptoms of intestinal irritation during the first week, it is extremely difficult to decide whether there is really a causal connection. It is a fact that many children with severe dyspepsia manifest no eruption, and that the latter—at least according to the author's experience—also occurs with very mild or no intestinal irritation. Absorption of enterotoxins might, of course, also take place. Leiner points out that the erythema may possibly be the first manifestation of an exudative diathesis, an unproved but very plausible theory, which might be explained by the frequent coincidence of severe symptoms of intestinal irritation. A certain similarity must be admitted with some fleeting forms of lichen urticatus, such as has been observed in a later period of infancy.

Intertrigo and Intertriginous Eruptions.

Intertrigo in the newly born occurs almost only in the genito-gluteal region; the other localizations in the axillary folds, neck, &c., are only seen exceptionally, but are found in a later period of infancy. In the newly born child intertrigo may be due to a certain disposition. The ultimate cause is almost invariably irritation from faeces and urine. The numerous (thin, slimy) stools of this first period of life, from about the second half of the first week, are a frequent cause for the intertriginous conditions, redness and wetting, and on prophylactic grounds necessitate frequent drying and thorough cleaning. It is by no means always sufficient just to change and clean the children before feeding them. If even this minimum is not carried out and the napkins are spared, such as sometimes happens in lying-in institutions, it is a procedure that cannot be sufficiently censured; for apart from the unpleasantness to the child the wet surface, robbed of its upper layers of epidermis forms a most favourable portal for the exciting causes of infection.

The treatment for moist forms consists of painting with 5 per cent silver nitrate solution and then covering with a thin layer of powder (talcum venet., or 10 per cent. zinc oxide). For simple redness, powdering is sufficient. Ointments are not very suitable for the treatment of intertrigo, at least in the earlier stages. Better results are sometimes obtained with zinc ointment, which is thinly smeared on the affected parts; the latter are thereby protected from further moistening.

Those eruptions known under the name "*erythema glutale*"

are related to intertrigo. They are characterized by the appearance of papular and vesicular efflorescences and erosions in the region of the nates. There are three principal types of gluteal erythema, the simple, vesicular and papular erythema, to which erythema vacciniforme may be added (Leiner). These are not dermatoses which are confined to the first week of life. As in intertrigo insufficient care of the skin and irritation by mechanical and chemical agents are, aetiologically, the most important factors.

Erythrodermia (desquamativa) (Leiner) is not a disease belonging to the newborn period. It seldom begins before the end of the first month. The disease may be mentioned here, as one has often tried to connect it with dermatitis exfoliativa—certainly incorrectly. Erythrodermia desquamativa is a skin disease, which occurs in severe chronically ill wasted infants, and consists essentially of redness and desquamation of the skin. It is more closely connected with those skin affections described as eczema seborrhoeicum, provided one does not understand an eczema with seborrhoea, but a disease sui generis.

True eczema, in its various forms, does not, strictly speaking, occur in the newly born period.

Localized Changes in the Skin

Lever reports a case of typical herpes zoster in a child of four days. Kaib has described a case in a newborn infant wherein there were localized, multiple, grouped scars, symmetrically arranged in the area of the first and second branches of both trigeminal nerves. He considered them to be the remains of an intra-uterine herpes, a view in which Kocks concurred.

Several cases of "spontaneous gangrene" are recorded in the literature. In Richmond's case two dark-red patches were present at birth on the head and the back of the neck. These patches became gangrenous quickly and spread rapidly. It was regarded as trophic disturbance due to some central disease. Bowes has described a case in an infant seventeen days old, starting as a pustule in the lumbar region and then extending rapidly. These cases are probably of the nature of noma; but Durando Durante records two cases in newborn infants which ended fatally, and which he regards as Raynaud's gangrene. Haas has seen gangrene of the toes develop in four weeks in an infant who had cyanotic discoloration of one of the lower extremities at birth. Foltanek has described a case of gangrene of the right foot, which was observed on the eighteenth day, and which was obviously the result of an embolic process of unexplained origin (fig. 68). The case terminated in recovery after spontaneous amputation (v. also p. 329).

Duvernay records several cases of gangrenous lymphangitis of the scrotum. Lymphadenitis of the scrotum may arise directly from skin infection, or from sepsis introduced at the umbilicus (Andrei). At any rate, these diseases are undoubtedly of an infective nature.

(III) Parasitic and Bacterial Diseases of the Skin.

Mycotic diseases of the skin are obviously very rare in infants, because the opportunities for infection are seldom present.

A few cases of typical favus, confirmed by bacteriological examination, have been described in infants of a few days old up to a fortnight (Schleissner, Sprecher). The infection is derived from



FIG. 88.—*Sporisorium vagabundum*. Infant 13 days old. (Politzek's case.)

another person suffering from favus, or from clothes or bedding. If there is opportunity for infection it spreads quite easily. *Herpes tonsurans* has been observed in very young infants (Tsch). Scabies does not appear to affect newborn infants, at any rate not in a fully developed form. It is an undeniable fact that pediculi capitis may settle on the head of the very youngest individuals.

Beck describes *infantile mycotic erythema* as a condition which

occurs within the first few days or weeks of life, and which resembles gluteal erythema in certain respects, but differs from it as a clinical entity. The disease begins with small red patches, varying in size from a pin's head to a millet seed. They become covered with central scales, enlarge, and eventually coalesce to large plaques and flakes which are sharply marked off from the surrounding area by circinate edges. Mycelium and spores can be demonstrated microscopically in the scales. Leiner describes a similar case of mycotic erythema. Beck considers that fungus found by him is identical with *oidium albicans*. It is evident that its condition is analogous with the process described by Ibrahim as *oidiomycosis* of the skin. He was able to demonstrate the existence of the *Oidium albicans* in the purulent contents of the vesicles about the genitals of infants a few weeks old, who mostly had some coincident intertrigo. Further researches are required, in order to decide whether the infection, in these cases, comes from the mouth cavity, and whether it can develop on healthy skin or only on skin which has undergone some secondary change.

Gonorrhoeal exanthemata of the newborn have also been described. Paulsen was able to demonstrate gonococci in the papules and pustules of the skin (especially the scalp) of infants whose mothers were suffering from gonorrhoeal vaginitis.

Pustular folliculitis and *furunculosis*, when they occur in early infancy, are in no way different from the same affections in older infants.

The most important infective diseases of the skin among newborn infants are *erythematous dermatitis* and *pemphigus neonatorum*.

Pemphigus Neonatorum and Allied Diseases.

The term *pemphigus neonatorum* embraces a group of skin diseases of infective origin, whose specific character consists of an eruption of vesicles. The anatomical structure of the skin of newborn infants seems to be well adapted for the development of manifestations of pemphigus; there is no period of life when exanthemata are so much associated with vesicle formation as early infancy. The wall of the vesicle consists of thin fragile epidermis, and the vesicle contains more or less thin liquid material. After the rupture of the vesicle the contents flow out, and the red moist corium is exposed. These characteristics, which are common to the various forms of pemphigus, are subject to considerable variation. The lesions are of the size of a hemp seed or a pea or lentil, but often much larger. The vesicles may be tense or flaccid. The fluid within them is either serous or opalescent or definitely purulent, and often contains blood. The skin on which they are situated may be diffusely inflamed, or they may be disseminated on areas of skin which are quite normal. They may arise on infiltrated or merely hyperæmic skin; but sometimes they appear to develop on absolutely normal skin. Sometimes the vesicles are sharply circumscribed; but occa-

sionally they spread superficially and lead to considerable peeling of the epidermis, so that the lesions lose their characteristics as vesicles. This explains the multiform clinical pictures of the disease which may differ considerably from one another.

The pemphigoid group of diseases may be divided into two classes: (1) An exogenous class which is caused by an infection of the skin, and (2) an endogenous class, which is the expression of some localized infection within the body or of some toxic process. Clinically, pemphigus may be divided into a malignant and a benign form; the endogenous class is obviously the more serious variety. It is sometimes very difficult to differentiate all the various clinical forms of pemphigus. It is very easy to distinguish a well-defined syphilitic pemphigus, or a pemphigus eruption in the course of definite septic diseases, more especially if the eruption is hemorrhagic or if other characteristic lesions occur in addition to the vesicles. Jarisch groups all these forms of disease together under the term "acute pemphigus," which he contrasts with "pemphigus neonatorum."

(1.) Simple Pemphigus Neonatorum.

(P. CONTAGIOSUS.)

The clinical picture of a mild pemphigus neonatorum is very characteristic in typical cases. A series of vesicles, either isolated or irregularly distributed in varying amount, appears upon skin which is otherwise unaffected. Their average size is that of a pea or hazel-nut, seldom larger. They are round or oval, and are usually surrounded by a delicate, narrow red areola. The palms of the hands and the soles of the feet which are the favourite sites of syphilitic pemphigus are usually, but not always, unaffected (Hagenbach-Burckhardt). The smaller vesicles are generally tense, the larger ones are often flaccid. Their contents are not, as a rule, clear like the serum in a vesicle from a scald, but rather opalescent and starchy. The vesicles of pemphigus do not, however, contain thick, definitely purulent material. The delicate vesicular membrane is often torn through in some of the lesions. If the contents have escaped, a moist or dried red plaque replaces the vesicle, and this is surrounded by shreds of moist or dry epidermis, which represent the remains of the vesicular wall. The lesions are seen in all stages of development, from the primary condition of a red patch, entire and ruptured vesicles, to raw areas where new epithelium is forming. It is a characteristic peculiarity of this disease that the lesions appear in crops. The mucous membranes are usually unaffected; but occasionally vesicles are seen on the red part of the lips, or red patches on the buccal mucous membrane (Bohn).

As a rule the infants suffer no constitutional disturbance. They put on weight and take their food quite normally, and they

have no fever. The ordinary form of pemphigus neonatorum appears to be attended by no discomfort.

The first signs of the disease usually occur in the second half of the first week—either in sporadic or epidemic form. In some cases the disease terminates in a few days after the appearance of a few vesicles. In other cases the disease is prolonged for two to three weeks, owing to the extension of the process and the occurrence of fresh crops. The outcome of the disease is always favourable, even if its duration is protracted, as long as the infants are otherwise well. Occasionally there is not more than one isolated pemphigus vesicle, so that one can hardly call the condition a disease.

As very similar clinical pictures manifest themselves in later infancy, Escherich has rightly proposed that the term *pemphigus infanthum* be substituted for *pemphigus neonatorum*.

The *Staphylococcus pyogenes aureus* is the organism most frequently found in the contents of the pemphigus vesicle (Almquist, Kowintzki, &c.). The presence of diplococci (Pasini) or streptococci (Vallois) must be regarded as very exceptional. Mild pemphigus neonatorum is closely related to impetigo contagiosa. If an adult is infected by a case of pemphigus neonatorum, it usually appears in the form of impetigo contagiosa, and on the other hand, if the mother has impetigo contagiosa and infects her infant, the latter will present vesicles of pemphigus. "As the histological findings are identical in both diseases, and as similar organisms indistinguishable from each other are found to be the cause of both, it is justifiable to assume that pemphigus neonatorum and impetigo contagiosa are essentially of the same nature" (Matzenauer). The difference between the appearances of the disease in infants and in adults is to be explained by the varying resistance of the human skin at different ages, and also possibly by individual idiosyncrasy. It is but seldom that the transmission of pemphigus to adults (e.g., to the breast of nursing mothers) is followed by the development of typical pemphigus vesicles. But not all authors recognize the identity of impetigo contagiosa and pemphigus neonatorum.

As a local infective disease of the cutaneous surface of the body, simple pemphigus almost always appears a few days after birth, if an opportunity for infection has occurred. Still some cases have been recorded wherein children have been born with pemphigus vesicles. Labhard and Wallart have collected sixteen cases of congenital pemphigus from the literature, and to these may be added the more recent observations of Cathala, Bar and Koblanek. Syphilis was definitely absent in all these cases. Three cases (Wickham-Legg and Hersch) are probably not to be classified as pemphigus, because they were obviously cases of hereditary epidermolysis bullosa; the duration of the disease in these cases was a matter of years. The cases of congenital pemphigus hitherto recorded have almost always run a favourable course and the

vesicles, of varying size, with which the infant is born, subside within a few days. The origin of these congenital lesions is not quite clear. It is stated that the mothers were in good health, so that one cannot assume that there was an intra-uterine infection from the placenta. Infection from the vagina could only occur if there had been a premature escape of the amniotic fluid (Koblanck). Occasionally, however, healing lesions have been found, on an infant just born, which is only possible if the infection has occurred before the rupture of the foetal membranes.

Blumenthal has recorded a peculiar case of a congenital hollow eruption, which was probably due to atresia, for the mother was treated with atoxyl a short time before her confinement.

Hereditary epidermolysis bullosa, which has been mentioned above, often runs in families, and some of the cases are congenital, according to Kanikv and Sutton, who suggest the name of congenital epidermolysis bullosa. The following are the chief signs. (1) Vesicles of varying size break out on different parts of the body, after mechanical injury, but not after other causes. (2) The vesicles are often hemorrhagic. (3) The nails appear to be affected by similar factors and become deformed or even destroyed. (4) The general condition remains good. In the case described by the above-mentioned authors, the first vesicles appeared on the second day of life.

(2.) Severe Forms of Pemphigus Neonatorum.

(P. NEONATORUM MALIGNUS.)

Although most forms of pemphigus are mild, there are some cases which are exceedingly severe and thus differ from the ordinary clinical picture. Any kind of pus infection in weak and debilitated infants may lead to serious consequences owing to generalized sepsis. This condition may develop from a local skin lesion in the form of a simple pemphigus, which is otherwise quite mild. But as a rule the condition of the skin is different in the cases which run a severe course. The vesicles, which are sharply circumscribed in the mild form and are seldom larger than a hazel-nut, increase rapidly in number and in size, extend over the surface and assume irregular shapes (fig. 69). The portions of the skin which are free from vesicles often undergo exfoliation of the upper layers of the epidermis, or these latter become remarkably loose. If one rubs the finger along the apparently healthy skin, layers of epidermis are easily pulled off. This loosening of the upper layers of the epidermis is called *epidermolysis*, and the case is termed *exfoliative* or *foliaceous pemphigus*. If this process extends, large areas of skin may become denuded of epidermis and the appearance resembles the condition of the skin after a severe scalding. In such cases the clinical picture is very like that of exfoliative dermatitis. Symptoms of generalized sepsis may appear coincidently with the

extension of the skin disease. It is then often impossible to decide whether a general infection of the skin is present or a secondary, septic eruption which is to be regarded as the expression of an infectious disease due to some other cause.

The course of malignant pemphigus is not always the same. Sometimes the disease lasts for days and sometimes weeks, until the children succumb to marasmus and chronic sepsis. The course, however, may be fulminating and lead to death within a few days. Finally, pemphigus appearing to be at first malignant, may come to a standstill and be cured.

Those diseases belonging to the class of pemphigus neonatorum, the mild as well as the severe forms, occur sometimes sporadically, sometimes in groups, sometimes even in epidemics. This is not extraordinary when one considers the presence of pus



FIG. 59.—*Pemphigus neonatorum* (severe form).

cocci in the contents of the bullae. In common pemphigus benignus, which also bears the name pemphigus contagiosus, it usually occurs in mild epidemics in lying-in institutes and foundling hospitals or in the practice of a midwife. It is probably the hand of the nurse that carries the infection. Children are, as a rule, very susceptible to pemphigus diseases. Transmission to adults occurs much less frequently. As already mentioned with the latter the infection generally takes the form of impetigo contagiosa. But occasionally, even in adults, one sees a pronounced formation of vesicles, especially surrounding the nipples, but sometimes in other parts of the body (Ostermayer). The source of infection might occasionally be the lochial secretion containing staphylococci, but owing to the ubiquity of staphylococci this cannot be maintained with certainty. There may possibly be a specific strain of staphylococci which causes the disease. The varying character of the genus epidemicus favours such a theory, as it is expressed both in mild and severe epidemics.



FIG. 30.—*Desmoglea nectatrix* in a breast-fed child 12 days old.
(Commencement of the disease on the 3d day, death on the 13th day.)

(3) Dermatitis Exfoliativa.

The characteristic changes of the skin in dermatitis exfoliativa have been appropriately described by the name introduced by Ritter. The disease consists essentially of inflammation of the skin with exfoliation, whereas the formation of vesicles forms only a secondary symptom.

Typical exfoliative dermatitis usually begins at the end of the first week or the commencement of the second, but it may start within the first day or two. The disease generally begins on the face, at the angle of the mouth and under the chin, but it may begin elsewhere, e.g., the abdomen (Litten) or the genitals (Sorgente), &c. The first change consists of a diffuse bright reddening and swelling of the skin, which gradually spreads over the entire body. The skin of the affected part looks thicker and more transparent, as if the horny layer had been swollen and macerated by a long-applied moist and warm compress (Escherich). In this stage the appearance of the skin looks like erysipelas (Escherich). (Ritter called the disease, originally, dermatitis erysipelatoïde). The epidermis becomes loose from the corium, so that there is a vesicular bulging of the former; but in the typical development of the disease definitely circumscribed vesicles are not found. Large areas of superficial skin appeared to be soaked in serous fluid, and a condition of epidermolysis and exfoliation follows. If the oral mucous membrane is affected it becomes covered with a greyish-white deposit. The corneal epithelium may also be affected (Elliot).

The course of the disease varies, even in typical cases. It may spread over the entire body, so that the skin may hang in shreds within a day or two, exposing, almost everywhere, a very red or livid corium. Infants affected in this manner succumb in a few days (fig. 70). In other cases the course is slower; the disease may remain at a height for over a week, and then be arrested, to subside gradually and terminate in recovery. In the 207 cases collected by Ritter the mortality was only 50 per cent.

The definition of the term, dermatitis exfoliativa, has been for years the subject of animated discussion among paediatrists and dermatologists. Clinical observations show that there are difficulties in separating it from the physiological exfoliation of the epidermis and also from pemphigus. Kaposi looked upon exfoliative dermatitis merely as an exaggerated physiological exfoliation. It is quite probable that some of the peculiarities of this disease are due to the conditions which exist in the infant's skin at the time of exfoliation; but the reddening and swelling of the skin differentiate it from the physiological condition (Litten). It is much more difficult to separate it from pemphigus. Although it is easy to differentiate typical forms of pemphigus neonatorum and exfoliative dermatitis, there are so many transitional forms that many authors hold that no sharp line of demarcation can be maintained between the two forms of disease (P. Richter, Ostermayer,

Bloch, Knöpfelmacher and Leiner, Hedinger). The authors (Luithlen, Pick, Rille), who uphold the view that there is a definite difference between pemphigus and exfoliative dermatitis, point to the absence of any initial development of vesicles and to the characteristic onset with reddening and swelling of the skin. Whereas exudation is the most prominent sign in the course of pemphigus, exfoliative dermatitis is distinguished by exfoliation. The histological changes described in exfoliative dermatitis are as follows: Oedema of the papillary and sub-papillary tissue, infiltration of mast and spindle-shaped cells, and great dilatation of the vessels. These changes resemble closely those found in cases of pemphigus neonatorum. The essential difference consists of a greater proliferation in the rete Malpighi, which never occurs to any considerable extent in pemphigus (Luithlen, Bender, Hansteen); but the difference is merely quantitative.

But it is not only the existence of transitional forms which proves the close relationship between the two diseases. The experience of epidemics shows that not only may a pemphigus become converted into an exfoliative dermatitis, but that epidemics of the two diseases run coincidentally, that very many cases of exfoliative dermatitis terminate as typical pemphigus, and that if the disease is spread by a nurse, dermatitis and pemphigus may occur side by side (Ravogli). But it must not be forgotten that the conception of dermatitis exfoliativa has undergone some change with the lapse of time. To-day we understand by this term a very severe illness which often proves fatal; but this is not the view held by Ritter when he described his cases. Ritter and Bohn, who describe very similar cases, characterize the disease as one which runs its course without fever and without affecting the brain functions. In Ritter's cases death occurred mainly from secondary infections (furunculosis, phlegmon or pyoderma) or from the complication of pneumonia. Bohn asserts that "the disease accelerates inanition only in those poor specimens of infancy who have no power of resistance; otherwise death only occurs from inflammatory visceral disease, originating in quite different causes. The skin disease is remarkably independent of the condition of the rest of the body, and has no tendency to produce complications and always progresses favourably." If we compare this statement with the cases of Escherich which had irregular fever and ended fatally, and with the severe and acute cases described by Litten, Behrend and Finkelstein, it is obvious that different authors describe different diseases under the designation of dermatitis exfoliativa. For this reason, the value of the attempts to find an explanation of the disease by means of etiological and anatomical researches is greatly prejudiced. Hedinger says with perfect justice that in a certain stage of the method process it is often a matter of the personal equation of the observer, whether he calls it pemphigus neonatorum or exfoliative dermatitis.

Staphylococci are usually found in the skin in exfoliative dermatitis and generally in the blood also; but this does not explain the

peculiar character of the clinical picture. Escherich thinks that exfoliative dermatitis develops on a groundwork of general septic infection. Although we may not be sure where the infection enters, there is every probability of bacterial processes playing the most important part in exfoliative dermatitis. The disease may occur in epidemic form, combined with severe pemphigus. Lühlen thinks that toxic influences also play some part in the etiology of the disease. This view is supported by the observations of Finkelstein, who found the blood and the viscera sterile in two very severe fatal cases.

TREATMENT OF THE PEMPHIGUS GROUP OF DISEASES.

The definite infectivity of the skin diseases belonging to this group demands energetic prophylaxis. The rare occurrence of epidemics or even of sporadic cases in lying-in institutions, in contrast with their previous frequency, speaks well for the hygienic management of these institutions. It may not always be possible for an overworked staff to maintain strict asepsis in the nursing of newborn infants, but nevertheless the modern spirit which prevails in maternity institutes has done much to eradicate parasitic skin diseases in early infancy.

As nurses are able to transmit the infection, it is urgent that they should be scrupulously careful in the washing and disinfection of their hands. The affected infants should be isolated. If the mother is suckling her infant, care is required during the process to prevent direct infection—in fact, it may be advisable to feed the infant with the milk drawn off from the breast, in order to avoid direct contact. At any rate, the infants should be taken away from the mothers during the intervals of feeding.

The most important consideration in the treatment of pemphigus, as in furunculosis, is to prevent the extension of the process by auto-inoculation. The lesions must be isolated as far as possible and the healthy skin must be cleansed. In mild cases, it may suffice to put the infants in an astringent bath containing oak bark or in a disinfectant bath of potassium permanganate or caustic sublimate. After the bath a desiccative dusting powder should be applied, such as zinc or dermatol with talc. If the disease is widespread, the moist areas should be painted with a 5-to per cent. solution of silver nitrate, an excellent remedy in all moist skin eruptions of infants. Ballin recommends that a 10 per cent. solution of ichthargan be painted on the raw surfaces, after which a thin layer of cotton-wool should be applied, and the solution be again painted on over this. Healing takes place under this adhesive covering. Ointments are useful when there is much scale formation, e.g., ung. sulphur rubr.; soft pastes are also serviceable, e.g., zinc paste, sulphur and zinc paste with the addition of aluminium acetate. If the skin is very acutely inflamed, as occurs especially in exfoliative dermatitis, gauze compresses of aluminium acetate should be employed, as in acute

eczema or erysipelas (Langstein). When the inflammatory swelling has subsided somewhat, the moist areas are painted with nitrate of silver solution, the rest of the body being powdered and swathed in loose bandages to prevent the sore places sticking to the clothes. In severe cases measures must be adopted to prevent collapse; the feeding must be attended to, mother's milk being the best nutriment, and in any case the infant must get enough fluid.

(IV) Diseases characterized by Induration, Swelling and Œdema of the Subcutaneous Tissue.

(1.) Sclerema Neonatorum.

Two different forms of skin disorder are designated by the term sclerema. In typical cases these two varieties possess distinct characteristics: (1) sclerema adiposum (fatty sclerema), i.e., sclerema in the limited sense, and (2) sclerema œdematosum or sclerœdema.

(A) SCLEREMA ADIPOSUM.

Typical fatty sclerema consists of a diffuse induration of the skin, most frequently affecting the calves and the face. In severe cases it may extend over large areas of the body, involving the lower limbs, the gluteal region, the chest and upper limbs, but leaving free the palms of the hands, soles of the feet, scrotum and dorsum of the penis. The skin feels as if it were tightly stretched, like a mask, over the underlying soft parts with which it appears to be closely united; it is almost impossible to pick the skin up in folds. Pressure with the finger leaves no pitting. The surface of the skin is usually smooth, somewhat shiny and generally pale, and as the affected infants invariably suffer from icterus neonatorum at the same time, its colour is yellowish, but it often has a livid cyanotic hue. The parts of the body affected by the disease look atrophic, and are encased by the hard skin covering as if by a closely adherent wax mould. The parts are in a high state of hypertonicity, and mobility is restricted both in the limbs and in the face which assumes a fixed immobile expression. The tightly compressed lips and the hardening of the skin over the zygomatic arches sometimes suggest the aspect of tetanus. The body temperature is almost always below normal, even as low as 30°C . (86°F). A temperature of 22°C . (72.6°F) has been recorded. The infants look frozen with cold, so that the picture resembles somewhat that of rigor mortis.

Sclerema always affects premature, weak or diseased infants. They generally lie in a semi-conscious state and motionless. Respiration is slowed, and the heart-beats are faint and slow. A sudden outburst of a shrill cry is very characteristic (*cri de détresse*). There is great difficulty in feeding these infants, so that an

oesophageal tube is often necessary. They may lie in this condition for days, hovering between life and death until the end.

Sclerema is not always generalized. Often only the face and calves are affected, the rest of the body presenting little or no induration and tension. Very low temperatures are not present in all cases. The prognosis is not then so unfavorable, and something may be expected from treatment; but sclerema is always a bad sign even if only slightly developed.

Fatty sclerema usually affects very atrophic infants, or those suffering from acute diarrhoea and sickness and of shrivelled appearance, up to the age of 6 months; but it often comes on spontaneously in the newborn on the third or fourth day, as soon as the fluid lost from the system reaches a certain amount, which may also occur under normal conditions. Sclerema adiposum, in its fully developed form, is, however, a very rare disease.

The pathology of fatty sclerema is not yet clear. The post-mortem findings are the same in cases of sclerema neonatorum as in other infants who are premature or debilitated. Not infrequently, they are quite negative, as far as gross changes are concerned, except that atelectases have been found in almost all cases. The histological examination of the skin often shows that the fatty tissue is completely dried up and it may present the characteristics of stratin. According to Lundén, the histological examination proves negative, or it may present the picture of a cachectic skin. He explains the apparent increase of connective tissue by the close agglomeration of the connective tissue fibres and the decrease of fatty tissue, and regards the cachexia as responsible for the great cellular proliferation; but in his opinion the pathology of the disease does not lie in an increase of connective tissue nor in an absorption of fat. Mensi, on the other hand, suggests that fatty sclerema depends upon a metamorphosis of connective tissue, which, in its turn, depends upon some congenital predisposition. There is hardly any reliable evidence for the view that there is some causal connection between sclerema and disturbances of internal secretion (hypo- and dysthyroidism).

The low temperature and the loss of fluid are very important features of sclerema. The peculiar rigidity of the skin is probably due to changes in the fatty tissue. The chemical composition of the fatty tissue of young infants differs from that of older individuals, and it also has a comparatively high solidifying point. But there must be special circumstances, which are still obscure, to determine the induration of the skin, because sclerema only occurs in a comparatively small number of infants with a low temperature, and because fatty tissue does not solidify after death in the manner of sclerema. There can be no validity in the assumption that sclerema neonatorum is caused by the coagulation of albuminous bodies after high temperature, because the disease hardly ever supervenes after febrile conditions. There are some cases which come on after sepsis, suggesting an infective basis for the disease.

(b) SCLERÖDEMA.

Whereas fatty sclerema encloses the affected parts tightly and diminishes their volume, sclerödema is characterized by swelling and a great increase in the volume of the affected parts. The skin is inflexible in fatty sclerema and loses its elasticity; but in sclerödema the skin presents the characteristic peculiarities of *teidema*, for a definite impression can be made on it by the finger. There is good reason for not summarily terming the condition *oedema*, for the swelling has a peculiar doughy consistence, and although it is yielding there is an induration about it. The term *sclerödema* was suggested by Soltmann, and it expresses very happily the two essential characteristics of *oedema* and *induration*.

If the sclerödema comes on during the period of *erythema neonatorum* the affected skin is red or pale; but it may be livid or even have a marbled appearance. The jaundice which is usually present confers a yellow tint on the skin at the same time. Small punctiform hæmorrhages are often seen, but these have no connection with the disease. The lower limbs are the favourite sites for sclerödema, especially the dorsum of the foot and the calves; but the thighs may be affected and also the mons *venereis*. All the other parts of the body may be affected, including the palms and soles. Sometimes the disease is limited to the dorsum of the feet and the neighbourhood of the genitals; but scattered, sharply circumscribed areas are never found. If widespread, it hampers the power of movement, just as in fatty sclerema. The temperature is subnormal in sclerödema, but never becomes so low as in cases of fatty sclerema. Occasionally the temperature is normal, or even above normal, but the affected parts are usually quite cool in these cases. Sclerödema is apparently more frequent in winter than in summer.

Premature and pany infants are the usual victims of sclerödema just as of fatty sclerema; but apparently normal vigorous babies are not always immune. The changes usually appear between the second and fourth day, seldom later.

Fatty sclerema is usually looked upon as a symptom of debility or of some severe illness; but sclerödema is considered by many to be a disease *sui generis*, a view which does not seem well founded, from the standpoint of general pathology. The anatomical findings are not usually characteristic. Sclerödema is found in association with the most varied diseases, such as congenital morbus cordis, pulmonary atelectasis, pneumonia, bronchitis, in the course of hæmorrhagic or septic diseases, and sometimes it is present when the post-mortem reveals no other morbid condition. For this reason, Lühlfen holds that the question whether sclerödema is a disease *sui generis* must be answered in the negative. He remarks that the clinical picture of the disease can be evoked by all possible causes and maladies which can produce congestion in the circulation, damage of the vessel walls or serous effusion in the tissues. In his opinion, the most important causes of sclerödema are defective

development of the infant, a peculiar constitution of tissue, especially of the blood-vessels, which easily permits transudation from them. The cases which Lühlen examined had skins which were like those of seventh month fetuses. According to Mensi, the histological examination shows signs of atrophy in the skin, absence of the stratum granulosum, thickening of the cells and fibres of the dermis, dilatation of the blood-vessels and hæmorrhagic effusion into the subcutaneous tissue. The œdema is not confined to the skin and subcutaneous tissue, but also involves the musculature. In two cases described by Finkelstein, it was not a clear cedematous fluid, poor in albumin, which was found in the tissues, but an amber-yellow serum which gelatinized on warming; and a similar fluid was found in the peritoneal cavity. On microscopical examination of the muscles, the transverse striation could no longer be seen, and there was some cellular infiltration around the capillaries. Finkelstein suggests that a prolonged low temperature damages the endothelium of the vessels, and finally produces a kind of coagulative œdema. Effusions are sometimes found in the body cavities; hæmorrhage into the lungs and pleural cavities are mentioned as occasional complications. Hervieux and Esser have described cases wherein hæmorrhages were found in the lungs and numerous rents in the distended and engorged pulmonary veins, without any sign of inflammation. The symptoms during life were severe dyspnoea and blood-stained expectoration.

Sclerœdema must probably be regarded as originating from similar causes as other forms of œdema, but as having additional etiological factors in cold, and the peculiar constitution of the skin and the fatty tissue of infants. For this reason there are transitional forms between ordinary soft œdema and sclerœdema, and œdema, in somewhat older unhealthy infants, may sometimes suggest sclerœdema.

The prognosis of sclerœdema is usually doubtful. Death may occur a few hours or a few days after its onset, or may be delayed for one or two weeks. But nevertheless this disease is not so grave as fatty sclerema, for in slight cases sclerœdema may subside; indeed, complete recovery may occur even after its extension over the whole body (A. Bauer). During the subsidence of sclerœdema the skin becomes flaccid and puckered. The œdema of the muscles and the resistance of the deeper parts usually remain for some time longer. The condition then suggests fatty sclerema, but the deep œdema can usually be dispersed.

Finkelstein reports a hitherto undescribed condition like sclerœdema, resulting from the direct action of organisms of inflammation. He associates the condition with erysipelatous swellings, which are not uncommon. He observed the case of an infant, three weeks old, who had a fatal streptococcal infection probably arising from the mouth. Numerous areas like sclerœdema developed in this infant; they coalesced in a few days and the skin looked like a case of diffuse sclerœdema.

Although typical cases of fatty sclerema and sclerodema appear to be different conditions, occasionally cases occur wherein one is in doubt as to which of the two conditions is present. Reaction to the pressure of the finger, which is generally regarded as the most important differential sign, may be of no help, for only a suggestion of pitting may be present, especially when the oedema is mainly in the muscles. For this reason many observers agree that combinations of both forms of oedema occur (Luitlén, Esch); it is also possible that the soft consistence of sclerodema may change later on into the hardness of sclerema (Griser). Clinically, it is justifiable to include both conditions under the term of sclerema, and to define it with Soltmann as "an induration of the skin and underlying tissue, with or without subcutaneous infiltration of serum, associated with a great fall in the body temperature."

Owing to the depression of temperature it is essential that infants with sclerema should be kept warm by means of hot-water bottles and hot baths. Tibone recommends that the affected limbs should be wrapped in cotton-wool, completely covered with gummed impermeable paper and fixed with bandages. Dufour wraps the infants in gummed plaster. The affected parts of the skin should be rubbed with warm oil to stimulate the circulation. Massage is most desirable in sclerodema and should be done several times daily in a centripetal direction, and thorough passive movements should be performed. Stimulants should be given preferably by hypodermic injection. Bauer recommends 4-6 grammes of brandy daily, but alcohol should be avoided, if possible, as it is a dangerous drug for a newborn infant. The general management and feeding must be conducted on the lines which apply to premature and debilitated infants.

(2) Scleroderma.

The nature of scleroderma is quite different to that of sclerema neonatorum, but the two conditions are no doubt frequently confused in medical literature.

Genuine scleroderma is a rare disease of infancy, but when it does occur, it is generally within the first three weeks of life. Of the cases hitherto reported some have been described as scleroderma (Cruse, Neumann, Haushalter and Spillmann) and some as sclerema. Luitlén has collected five such cases from the literature (Money, Barrs, Garrod, Blocker, Bunch). It is possible that the cases described as "sclerema" by Waterhouse and Carpenter and Sheffield Neave were really cases of scleroderma. In both cases there were patchy sclerematous changes present, in addition to a diffuse sclerema-like constitution of the skin; in one case the disease gave the impression of sclerema, but developed into disseminated islands of sclerema.

In the rare instances of typical scleroderma in infancy, the disease began within the second or third week of life; it has, however, been observed immediately after birth, and it seems chiefly to

attack infants who are well nourished. It does not begin on the peripheral portions of the body like sclerema, nor does it usually start as a diffuse infiltration, but it occurs in localized areas as hard infiltrations definitely marked off from the surrounding skin which retains its normal suppleness. The infiltrated areas are usually of the size of a lentil seed, but they may spread and involve extensive surfaces, though it remains possible to recognize the small areas in which the large patches began. The limits are sharply defined, and the edges are jagged or sinuous. The colour of the diseased areas is usually reddish or livid. Sometimes the skin is sensitive when the disease begins, but its mobility remains unaffected. At first new areas of disease appear in different parts, and then the induration begins to subside. After a few weeks, or, at most after a few months, a perfect recovery ensues. Cruise, however, described one case wherein puckering persisted in the areas of the skin thickening with signs of atrophy, similar to the usual results of scleroderma in adult life.

The treatment consists of warm baths, which tend to soften the indurations, also rubbing with oil, iodine vasogen, or mercurial ointments; but as the prognosis is always favourable, and as there are no subjective symptoms, treatment is somewhat superfluous.

(3.) **Edema of the Genital Organs**

The edema described under this head, occurring in infants within the first days of life, comprises several varieties which are quite different clinically and etiologically.

More or less marked swelling and usually slight redness of the labia majora are very frequent, and to some extent are present in almost all newborn female infants. The labia often seem to be infiltrated with serous fluid. In other cases they present a well-marked, tense, oedematous swelling. This edema of the vulva, which usually disappears more or less completely in a few days, is regarded by many as a congestive edema due to pressure during birth (Finkelstein), while others regard it as a symptom of frequency in Halliun's sense (Zappert).

Much more rarely than in girls, corresponding swellings of the penis and scrotum are found in boys.

Very frequently in both sexes the skin of the mons veneris exhibits a swelling, which in its consistency resembles sclerodema.

All the above conditions are of a harmless nature.

The skin affection described as chronic idiopathic genital edema, attacks boys almost exclusively. It manifests itself in oedematous swelling of the mons veneris and the genitals, sometimes also the parts bordering the thighs. The oedematous region of the skin is sharply defined above by a horizontal line half way between the navel and the symphysis. The skin shows no signs of inflammation, it is pale and of a normal temperature. The edema generally appears 1 to 3 weeks after birth, rarely earlier, and generally remains unchanged for some time, often months, without giving the child

any trouble, and then gradually disappears. The prognosis appears to be entirely favourable, as such forms of oedema are hardly ever observed in children after six months. Frieljung, who has given his attention to this not uncommon disease, considers it to be the result of a very mild infection proceeding from the navel, analogous to erysipelas, but which leads to a torpid oedema. He supports his theory on the above-mentioned findings of Finkelstein, who showed that streptococci were present in a case of genital oedema resembling sclerodema. Possibly also local conditions of irritation of the genitals also play an aetiological part, such as phimosis, preputial concretions, &c. (Neurath). It is possible that a too tight umbilical bandage may cause oedema of the abdominal regions, but such swellings soon disappear after the removal of the obstruction. The above-mentioned oedemata are not influenced by treatment. Compresses are almost useless.

Probably connected with the above-mentioned genital oedema there is a case Petrone described as "oedema chronicum congenitum," in which there was swelling of both lower extremities and scrotum, which subsided in the course of six months without disappearing completely, as a swelling ranging in intensity remained on the dorsum of foot. In this respect the case resembles those diseases which have been described as acute circumscribed oedema (acute angioneurotic oedema, Quincke's disease). The latter occur, as a rule, at a later period of life; the youngest case described by Kassirer was that of a child of one and a half months. A case described by Sutherland is probably an example of the kind (from birth oedema of the feet, varying in intensity, and pinkish-red colour of the soles, which become intensely blue when cold; also frequent eruptions of lichen urticatus).

(4) Rarer Forms of Oedema.

GENERAL IDIOPATHIC OEDEMA.

During the newly born period and under normal conditions the pathologically increased disposition to retention of water is very seldom observed in a marked degree. One frequently has occasion to notice this retention in sickly infants, especially after administration of food relatively rich in salt. It manifests itself sometimes merely in abnormal increase of weight according to the amount of salt in the food and the severity of the existing disturbance of nutrition, and sometimes in the appearance of pronounced oedema. The healthy infant also exhibits to a certain degree a weakness of "salt excretory function" and all the more so the younger it is (Finkelstein); but under the usual conditions of nourishment this functional retention in the regulation of water and salt excretion is seldom seen in the newborn child. It only occurs if the water or supply of salt exceeds a certain measure. Birk reports the case of a healthier child, weighing $3\frac{1}{2}$ kg., that with minimal nourishment, but ample supply of fluid (up to 530 g. wa), showed a normal weight

curve, but after ten days manifested oedema of the back of the hands and feet; it disappeared as soon as the child was given the same amount of mother's milk instead of the tea.

From the above two curves (figs. 71 and 72) it may be seen that in the newborn child during the first few days of life the weight curve may be noticeably altered by the amount of salt solution given, in the sense of water retention. Increase of weight and oedema occur still more easily in debilitated or sick newborn children after administration of salt and water. This may be noticed in those children who have been given relatively large quantities of normal saline solution by mouth or subcutaneous injection to prevent exsiccation.

Both curves show the course of weight of two children, who were given 4 per cent. salt solution (NaCl , CaCl_2 , KCl , NaHCO_3 in proportion to Ringer's solution). Both have a perfectly normal course. (The marks denote that too g. 4 per cent. salt tea was administered to each.)

The first case (fig. 71) was that of a child that drank only small quantities from a breast containing very little milk. On the second and third day each quantity of milk weighed no more than 5–10 g., and frequently could not be weighed at all. But in spite of this the weight curve, under the influence of the salt tea, fell by merely 120 g. Up to the seventh day the quantities of



FIG. 71.

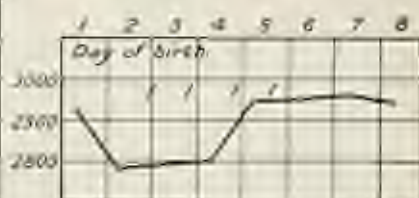


FIG. 72.

milk did not exceed 20 g., yet on this day the birth weight was attained. In the other case (fig. 72) the feeding conditions were favourable. On the fifth day the quantities weighed 70 g., on the sixth 100 g. The influence of the salt tea cannot be underestimated; the maximum weight occurs on the second day, the birth weight is attained on the fifth day.

LYMPHANGIECTATIC OEDEMA.

Fromme gives this name to a case of oedematous swelling of the hands and feet (including the palms, soles and the leg). At the first the swelling pitted on pressure, but subsequently it assumed the consistence of dough. Later on the oedema subsided in one hand, but the skin remained too loose for the hand. Similar looseness of the skin existed at birth, over the chest and back, especially at the back of the neck, where the skin could be lifted up in large folds like the skin of a young dog. Fromme assumes, therefore, that oedema had also occurred before birth. His opinion is that the subcutaneous lymph spaces were unusually well developed and that this led to more or less pronounced stagnation of lymph. Veit has described an analogous case. Two similar cases are recorded by Finkelstein; in one case there was extreme congenital oedema of the

left arm and shoulder extending to the finger tips, especially when the arm was in a dependent position. The oedema gradually diminished and disappeared during the second year. In the other case there was slight oedema of both upper arms, and the skin was remarkably flaccid; these symptoms also disappeared towards the end of the second year.

CONGENITAL GENERAL OEDEMA.

(*Hydrops foetus universalis*.)

Some of the cases of generalized oedema of the newborn, described in the literature, must be attributed to diseases of the heart or kidneys. But besides these, there are some cases of congenital oedema which are undoubtedly due to some other common cause. These cases are more interesting from the pathological than from the clinical aspect, because they consist mostly of premature infants, who are born dead or who die a few minutes after birth. The entire skin presents a more or less pronounced oedematous swelling which often attains massive proportions, and there are also serous effusions into the body cavities (Broekhuizen).

The most characteristic feature of the post-mortem findings is said to be an enlargement of the spleen. Histological examinations reveal the presence of well-developed areas of haematopoiesis in the liver, kidneys and spleen. The blood picture is very striking, containing numerous erythroblasts and myeloblasts or large lymphoid cells, which Rautmann terms lymphoid basophile mother-cells of the erythroblasts. These changes are regarded as the expression of a pathological irritation of the blood-forming organs (Schröder, W. Fischer). The cause of the disease is unknown; but it is not syphilis. The fact that most of the infants were born of mothers who suffered from typical nephritis of pregnancy renders it very probable that "some obscure metabolic product of the mother is the responsible cause." B. Wolff observed that fetuses developed general oedema and ascites after the removal of the maternal kidneys.

Cozzolino also attributes his case to toxic causes; the mother of the oedematous infant had been suffering from nephritis during the later months of pregnancy and the puerperal period. The infant was breast-fed, and oedema appeared on the eyelids and back of the hands on the fourth day; it gradually extended over the whole body, but completely disappeared in the course of four weeks, when the symptoms of nephritis in the mother also vanished. Renal oedema is very rare in the newborn infant, because nephritis is very rare.

(5) Congenital Elephantiasis.

This term embraces many congenital changes, varying in their anatomy and aetiology, the common factor in them all being a diffuse thickening of the soft parts in certain portions of the body, more especially the limbs.

It is necessary to distinguish teleangiectatic elephantiasis from the lymphangiectatic form, these conditions being due to abnormalities in the vascular or lymphatic system (cystic dilatation, &c.). Some forms of elephantiasis are probably caused by the congestion which follows intra-uterine constriction by amniotic bands, and in these cases transverse furrows mark off the elephantiasis portions from the healthy parts. Some authors (Nelsen, Bernhard and Blumenthal) think that these furrows are caused by thick fibres binding the subcutaneous tissue down to deeper parts, these fibres running transversely at the border of the joint.

In other cases there appears to be a simple hyperplasia of the connective tissue, due to some morbid predisposition or inherent tissue defect (Nöggerath). This view is strengthened by the occurrence of elephantiasis in families and by its association with other malformations in the same infant (Nonne). The forms of diffuse simple elephantiasis which manifest themselves as a cushion-like bulging of the affected soft parts depend upon a predisposition to the new formation of connective tissue, which also leads to the development of circumscribed forms of lobular elephantiasis and fibromatosis multiplex, &c. (Spietschka, Mainzer, Helbing, Behan). The skeleton may also be involved in elephantiasis (Rach).

The prognosis is unfavourable as far as recovery goes, because the condition is really a malformation; but the general health of the infant does not suffer. The affected parts have a tendency to erysipelatous inflammation, and this may be a source of danger (Virchow). Treatment is of little avail; massage and bandaging are useless. In later life the question of surgical methods may be considered.

CHAPTER XI.

THE UMBILICUS.

(A) The Umbilical Cord and its Treatment.

Since the introduction of aseptic methods in midwifery, the obstetrician has shown considerable interest in the treatment of the umbilical cord. There is no doubt that the methods have multiplied enormously, and many are merely unnecessary modifications. Of course, the umbilicus is a portal of entry of infection in the infant, but it must not be forgotten that there are other ways by which infection can take place, and the umbilicus must not claim exclusive attention from this standpoint. Nevertheless it is satisfactory that the umbilical cord receives careful attention, because umbilical infection is now very much rarer than it was in pre-aseptic days. The severe and fatal forms of umbilical disease and umbilical sepsis are now extremely rare, especially in lying-in hospitals.

In the treatment of the umbilical cord three points must be considered: (1) its separation; (2) care of the umbilical stump; (3) the

umbilical wound after the stump has fallen off. The cord is usually ligatured in two places, 5-10 minutes after the birth, a few centimetres from its insertion into the abdomen. One ligature is applied at about the distance of three finger-breadths from the umbilicus, and the other about two finger-breadths further on. The material employed is usually sterilized linen tape $\frac{1}{2}$ cm. wide, which has been soaked in a sublimate solution. The cord is cut with a sterilized scissors between the two ligatures. In order to prevent recurrent hæmorrhage from the stump, Seitz advises an additional ligature closer to the umbilicus. It matters not whether the ligatures are applied a little further from or nearer to the umbilicus (Döderlein's clinic 10·7 cm., Küstner's clinic, Seitz 7·2 cm.). Runge applies only one ligature and cuts the cord on its distal side. Rothschild recommends elastic ligatures with india-rubber tapes which are drawn through a metallic ring.

There has been much discussion as to the correct time for separating the cord; but it appears that the additional current of blood flowing to the infant, if the separation of the cord is delayed, is of no significance (Runge). Czerny-Keller state that there is no reliable evidence to show that the time of the separation of the cord has any influence on the nutrition, metabolism or development of the infant. A compromise is usually adopted, in that the cord is not tied immediately after birth, nor does one wait till the expression of the placenta, but only until the cessation of pulsation in the cord, an average of five to ten minutes.

After the separation of the cord a portion of it still remains attached to the body of the infant. This may be permitted to dry up under an appropriate dressing. It has been proposed, of late, that the cord should be still further shortened, which can best be done after the first bath. The cord is again tied just at the umbilical ring or half a centimetre from it with linen tape (Altfeld), thread (Martin, Weckerling, Stolz), or with catgut (Leithe), and it is then cut through with scissors. Martin suggests the use of red-hot cautery scissors to produce a slough (Rieck). Instead of the ligature, the stump of the cord can be secured by an appropriate instrument. Bar and Penndecerd apply a clamp quite close to the umbilical ring, which they allow to remain for twenty-four to forty-eight hours after the stump of the cord has been cut off (Orlowsky). Pinard, Ponak, Gauss, Gigli and Schlank use similar clamps. The method practised in Schauta's clinic is very useful (Feitler); the cord is seized by a sterilized hæmostatic forceps, cleansed with alcohol, just at the edge of the skin, and is then cut off with a scalpel or blunt-pointed knife immediately above the instrument, which is allowed to remain on for at least ten minutes; a thin membrane is left no thicker than paper. Marcus recommends the application of a small clamp for twelve to twenty-four hours, to the umbilical stump around which silver foil is placed; a thin layer of cotton-wool is put above and below the clamp and a bandage over this; the umbilical stump is as thin as paper and is closed with the silver foil as with a plug.

The only risk attending the cutting-short of the umbilical cord is that of hæmorrhage, which may be difficult to arrest in the absence of an adequate stump; but experience shows that not much danger is to be apprehended. In most cases there is no bleeding after the removal of the clamp, even without any ligature, or if there be any bleeding it is merely parenchymatous and stops spontaneously. It is obviously necessary to be sure that there is no hæmorrhage from the vessels which have been cut through, before applying a dressing; should this be the case a ligature will be required. In order to avoid the chance of hæmorrhage absolutely, both a clamp and a ligature may be employed (Jägeroo).

The absence of a long umbilical stump facilitates the treatment of the wound by asepsis and drying. Before applying the binder, the cord and the surrounding skin should be washed with a swab soaked in 90 per cent. alcohol, and the wound and the cord should be dusted with dermatol or similar powder. Some authorities dispense with the washing and powdering but put the binder on at once (Range). If a long piece of cord remains it should be wrapped in sterile wool or gauze; if it has been cut quite short, a piece of sterile gauze or wool is put directly over the umbilical wound and the binder is applied over this.

Dermatol is the best dressing powder to apply to the umbilical stump to ensure rapid drying and the prevention of bacterial activity; but xeroform, aërol, vioform or salicylic acid powder may be used instead. Leubie recommends diachylon powder. Recently, very good results have been obtained with "bolus alba," which has energetic desiccating properties and has the advantage of cheapness (Horn, Galatti). The bolus must be thoroughly sterilized by heating up to 170° to 200° C. Zweifel has observed four cases of tetanus following the application of this bolus, owing to the neglect of sterilization. Petermüller recommends dry kieselguhr (precipitated calcined siliceous earth), which can be annealed without fusing. Geszner recommends applying a ligature at the umbilical ring of tape soaked in formalin, after the cord has been separated in the ordinary manner; the cord is then cut off to within 1 cm. and is painted daily with formalin, a preparation which is especially suitable for the cord owing to its bactericidal and hardening properties.

The umbilical dressing should be a hand-breadth in size and may consist of calico, gauze or flannel, and is applied around the cord which is previously covered with sterile gauze. This dressing has the disadvantage of becoming easily displaced upwards or downwards, so that the umbilical wound is exposed. For this reason the application of light elastic bandages is recommended (Tetra), care, of course, being taken that the bandage is not too tight. An umbilical bandage, made too tight, is highly unpleasant for the child, and not infrequently the cause of inexplicable crying.

Flick's apron bandage (fig. 73) is much used and highly to be recommended. A soft muslin bandage, about 10 cm. wide, with woven edges, is sewn four-fold together into a square piece. At

the upper and lower side two soft muslin tapes 110 cm. long, 1.5 cm. wide, are sewn, so that right and left and above and below a free end of tape, 50 cm. long, is left over. A third tape, 60 cm. long, is sewn vertically to the others on the left side of the square (Eischerich). The sterilized apron is placed over the navel, the side tapes are slung round the body, then brought forward and fastened over the apron after they have been drawn through a loop made at each edge. The upper tape is hung round the child's neck and fastened on the other side. With boys, in order to avoid wetting, a piece of Billroth muslin about 10 cm. wide and 12 to 15 cm. long can be laid under the square over the symphysis and genitals. The umbilical bandage does not become pushed out of place and can be left when inspecting the navel; only the lower end need be loosened and the apron folded upwards.

Vomel recommends the following method: a round compress about 6 cm. in diameter, made of four-fold not too closely woven muslin, and impregnated with dermatol, is so fastened by means of a plaster ring $5\frac{1}{2}$ cm. internal and 7 cm. external diameter, that the compress forms a little bag to enclose the remainder of the umbilical cord.

The umbilical bandage is left by some till the fourth or fifth day (Eicke), sometimes during the whole first week, without being changed, until the cord falls off (Ahlfeld, Petenmöller). This is quite possible with a well-fixed bandage. But as a rule, owing to wetting of the bandage (which cannot altogether be avoided, even with an impermeable material), it may have to be changed. Also for the sake of precaution it may be necessary to inspect the umbilical wound daily, and possibly powder it and cover it with a sterile piece of gauze.

It has been much discussed whether, in the interest of rapid drying and aseptic healing of the wound, the child should be bathed daily or not (see Hartz). The majority of authorities is in favour of omitting the daily bath and substituting for it daily ablutions till the cord has fallen off. The importance of this question might be overestimated. It can hardly be assumed that the ordinary daily bath and fresh bandage should be disadvantageous to the child. At the most it is possible, that with a long, gelatinous cord mummification might be less rapid; but this can be aided by application of drying powder. Therefore the daily bath has not lost all its adherents (Wolde). In lying-in institutions it is certainly advisable to omit the bath in order to save time. With the hard work imposed on the staff, it is unfortunately impossible to guarantee the bath



FIG. 73.—Eicke's apron bandage.

being correctly given, and in view of this it is better for the nurses to consider the infant's navel as a *non me tangere* and to confine themselves at first to sufficient washing of the body. Comparing 1,000 bathed and 1,000 unbathed children, Berend and Rácz came to the conclusion that the bath had no sort of influence on separation of the cord. But there were twice as many umbilical diseases in the bathed as in the unbathed; and also in the latter the affections were milder.

Under normal conditions the remainder of the umbilical cord dries up and mummifies, either as a small appendix attached to the navel, or as a short stump in the umbilical region, without extending beyond the level of the latter, and gradually detaches itself from the umbilical base. The cord seldom falls off before the fifth day; as a rule it happens between that and the tenth day, and only exceptionally later. The various methods of dressing the navel have been estimated according to the time of the falling off of the cord, and the prize awarded to the method which statistically was found to produce its early separation. But that is hardly the most essential point. The chief object in dressing the navel must be to dry the stump as rapidly as possible, so that the separation provokes no inflammatory reaction, and that during and after the release of the cord there is no secretion, worth mentioning, coming from the navel. In short, the whole procedure should be carried out under aseptic conditions. Whether the remains of the cord separate a few days earlier or later cannot be of importance.

If the cord has fallen off, under normal conditions only a very small wound should remain, and soon disappear under the parts of skin which border it. Slight moisture during and after the release of the cord must not be considered as abnormal. In such cases the umbilical region should be lightly dabbed with 3 per cent. hydrogen peroxide. Until the final healing of the wound, the latter should be covered with sterile gauze, strewn with powder, and bandaged. Bandaging is necessary, as infections may easily occur just at this time. If the wound is healed, which is generally the case about two weeks after birth, further application of an umbilical bandage is unnecessary (see p. 276).

(B) Umbilical Diseases.

(1) Anomalies of the Umbilicus

UMBILICAL HERNIA.

Umbilical hernia denotes the presence of the contents of the abdominal cavity in a hernial sac consisting of peritoneum, amnion and a thin layer of Wharton's jelly, which corresponds to the hernial part of the umbilical cord (fig. 74). The hernial aperture is formed from that opening in the abdominal wall which under normal conditions narrows itself into the umbilical ring; in individual cases it varies in size. In extreme degrees of the anomaly

the greater part of the anterior abdominal wall is completely absent. In the majority of cases the contents of the hernial sac are formed of omentum, small intestine (ileum) and caecum, and frequently part of the liver; in big ruptures the large intestine, spleen and other abdominal viscera may also be included.

As regards the genesis of umbilical hernia according to Harnes three theories may be considered:—

(1) According to Ahlfeld there is arrest of development from abnormal adherence of the vitelline duct to the intestine. If the separation of the vitelline duct, which usually occurs at the end of the tenth fetal week, does not take place, the intestinal coils which were formerly in a physiological position cannot return into the



FIG. 74.—Umbilical hernia.

abdominal cavity, and the umbilical aperture is complete. The umbilical ring then either closes to a certain extent, or there remains a more or less large fissure in the anterior abdominal wall. Owing to pulling of the vitelline duct more intestinal loops and viscera may enter the hernial sac.

(2) Aschoff regards the primary cause of the anomaly as a persistence, in the human foetus, of the dorsal, concave curvature of the vertebral column. If there is no transition to the dorso-convex curvature, the result is a deficient closure of the abdominal walls. The liver does not develop as normally at the side of the anterior abdominal wall, but takes an abnormal position. Simultaneously anomalies in the hepatic vessels may be found.

(3) Kernerauer is of opinion that an arrest of growth of the primitive vertebra, very early, at the latest in the third fetal week,

is the real reason for the occurrence of these herniæ. According to this theory, there would be a primary disturbance in growth of the abdominal walls. The same explanation may possibly not meet all cases.

From a clinical standpoint two types of the anomaly may be distinguished:

(1) Ectopia of the abdominal viscera, or large herniæ, in which part of the anterior abdominal wall, varying in size, is altogether absent. In these cases an umbilical ring does not exist, the rectus muscles are generally not present. These forms are classified as abdominal fissures (*fissura abdominalis*); they may be combined with ectopia of the bladder, malformations in the genital regions, etc. (Steinbüchel). These herniæ exist at an early embryonal period, before the third month of pregnancy (Escher).

(2) True umbilical herniæ which occur when the navel is already formed. In these cases an umbilical ring is present, the rectus muscles are formed, but are generally diastatic.

The umbilical herniæ consist of semi-circular or longitudinal tumours of the umbilical region, and which are surrounded by a more or less transparent sheath, through which may be seen the contents of the hernial sac (intestinal loops, liver, &c.). The umbilical cord is situated close to the top of the tumour and generally in the region of its lower half. The thin sheath of the hernia is sharply defined against the skin of abdomen. Its base is sometimes broad, particularly in large ectopias of the abdominal viscera, sometimes so small that the tumour protrudes. The herniæ vary greatly in size; tumours of all sizes occur, from the size of a nut to that of a child's head. The thickness and resistance of a hernial sac also vary. Sometimes it is so delicate that it bursts at the time of delivery, and the abdominal viscera presents through the rent; in other cases it is entirely or partially firmly constructed.

If the herniæ are left to themselves, as in the case of the umbilical cord, necrosis or gangrene of the coverings and subsequent peritonitis may occur; the children succumb during the course of the first few days or weeks. But in rare cases—provided there is aseptic treatment—gradual recovery may occur. The contents of small ruptures may return into the abdominal cavity and the entrance be closed by granulations. In larger ruptures the entrance may be narrowed by granulations and skin healing proceeds from the peripherally situated abdominal wall, while the coverings of the rupture are cast off from the border (fig. 75). There generally remains a hernia of the linea alba with wide diastasis of the rectus muscles (Sittler).

With a relatively narrow hernial orifice symptoms of incarceration may occur (Seiffer, Maerklein, Hannes). At first they manifest themselves in disturbances of intestinal circulation, which may be recognized by marked injection of the intestinal loops shimmering through the sheath of the rupture.

In rare cases spontaneous recovery has been observed under dry treatment with dermatol or zinc oxide-dermatol powder (Sittler), and

with warm moist compresses with physiological salt or boracic solution (Durlacher). Nearly all authorities are nowadays of opinion that umbilical hernia, in all cases, should be operated on as soon as possible. Considering the severity of the affection the results of operations are very satisfactory; this may be gathered from the following table of Lotheisen (1903) dealing with ninety-one cases in the antiseptic period:—

| | Recovery | Mortality |
|------------------------|-----------------|-----------------|
| Conservative treatment | 34.75 per cent. | 65.25 per cent. |
| Operative treatment | 29.6 " " | 29.4 " " |



FIG. 75.—Spontaneous recovery of umbilical hernia.

As regards the prognostic importance of the time of operation it must be remarked that the sooner the operation the better the prognosis—if possible it should be undertaken during the first 6 to 12 hours—but that, on the other hand, treatment at a late stage should not be neglected for children, even when peritonitis is present. On the fifth day Rothe was able to cure a case by operation, although a gangrenous part of the liver in the rupture had to be removed; Zillner reports a similar case. Finsterer gives the following table:—

| Operated on:— | | | | Deaths | |
|-------------------------------------|---|---|----|------------|---|
| Up to 6 hours post partum 14 cases. | | | | 0 | |
| 0 to 12 | " | " | 7 | 1 | " |
| 12 to 24 | " | " | 12 | 5 | " |
| " 36 | " | " | 3 | 1 | " |
| " 48 | " | " | 4 | 2 | " |
| 5th day | " | " | 1 | (Rothe) 0 | " |
| 6th day | " | " | 1 | (Garzin) 0 | " |
| Date not given | " | " | 3 | 1 | " |
| 25 cases. | | | | 10 deaths. | |

Fiedler operated on a large umbilical hernia on the fifth day with satisfactory result; Huelst also saved a case by operation.

A case operated on by Bayer, a very large burst umbilical hernia, shows that an early operation is not hopeless, even in the severest cases; this case was operated on during the first hour of life and recovered completely (Piering). Marek also operated on a burst hernia with satisfactory result.

Of the various methods of operation, the radical one is generally chosen: Opening of the hernial sac; replacement of the contents; if necessary, fissure of the umbilical ring. This method is nowadays



FIG. 76.—Umbilical hernia.

preferred to the extra-peritoneal method of Olshausen. In the latter, amnion and gelatinous material are dissected from the peritoneum, the unopened hernial sac and contents replaced and the skin stitched over it (Ringel). In small ruptures the viscera can be replaced, according to Breus' method, without opening the hernial sac; through the skin a clamp is placed round the hernial sac, this is opened and removed, and two or more stitches passed beneath the clamp, according to the size of the rupture (percutaneous ligature). Replacement and application of a hernial truss are only suitable for very small hernias.

AMNION NAVEL AND CUTIS NAVEL.

Amnion navel is one of those rare and harmless anomalies caused by the amniotic sheath of the umbilical cord overlapping the abdominal skin (fig. 76). This amniotic mantle, which may attain the size of a five-shilling piece, shrivels up, and leaves a defect which gradually closes by granulation.

In the more frequent "cutis navel" the skin overlaps part of the umbilical cord (fig. 77). When the latter falls off there remains a small stump of skin, which shrinks in the course of further development, but sometimes remains permanently. Apart from this small cosmetic defect the anomaly is insignificant.



FIG. 77.—Cutis navel.

(2) Diseases of the Umbilical Cord.

ISPURMS.

Injuries to the umbilical cord are only of danger to the child if they are intra-uterine. Under certain circumstances the danger is equally great, whether the umbilical cord is completely torn, or whether the tear only affects the umbilical vessels, the amniotic sheath remaining uninjured; in the latter case the hematoma may lead to compression of the umbilical vessels and to interruption of circulation, as with hæmorrhage in the free amniotic cavity. The chances of preserving the child's life are greater the later the umbilical injury occurs and the quicker the birth.

Extra-uterine tearing of the umbilical cord occurs principally with precipitate labours. The weight of the child falling out with so much force is, as a rule, sufficient to cause a tearing of the umbilical cord. The strength of the cord varies considerably, although gelatinous, thick cords are as liable to tear as thin ones. Much twisted cords with varicose vessels burst relatively easily. The umbilical cord can also tear with a prostrate position of the uterus, if the child, through violent uterine contractions, is forced vehemently from the genital passages. Finally, umbilical rupture may occur as a result of obstetric manoeuvres. The tears may easily occur if the cord is too short or shortened by being twisted round the infant's body. They generally occur in the fetal part of the umbilical cord.

The dangers to the child are comparatively slight. As, under normal conditions, after separation of the cord and development of pulmonary circulation the arteries immediately contract, bleeding to death is hardly to be feared. There is no hæmorrhage from the umbilical veins after connection with the placenta has ceased. Hæmorrhages occur, as a rule, only in asphyxial conditions and other disturbances in the infantile circulation. Then they are more severe the nearer to the body the cord has been torn (J. Bayer).

In order to prevent hæmorrhage, a ruptured umbilical cord must always be ligatured. In lesions of the navel itself the hæmorrhage generally ceases after application of a tampon (possibly saturated with a hæmæstatic); ligatures are seldom necessary (Stöckel). (See also p. 496).

Hæmatomata of the umbilical cord may, as already mentioned, be intra-uterine, e.g., above a twisted part (Bussmann), or they arise during birth from overstretching or from mechanical pulling. The usual cause of hæmatoma is bursting of a varicose part of the umbilical vein. The remnant of the cord with a hæmatoma appears as a reddish or blue-black, thick, sausage-shaped structure, occasionally the size of a hen's egg. The hæmorrhage is of no importance for the child. One can either await the spontaneous falling off of the cord, or tie off the infiltrated part and remove it, which is more to be recommended in order to prevent secondary infection (Beissard and Roche, v. Westphalen, Couvelaire, Krömer).

TUMOURS.

There are cystic and solid tumours of the umbilical cord. The former are, as a rule, the size of a pea, and may in rare cases attain the size of a child's head. They are probably derived from the allantois, but possibly from persistence of the remains of the embryonal peritoneal canal (Laçouture). Teratoid cystic tumours are also supposed to occur. Stolz describes a case of umbilical cord cysts with simultaneous umbilical hernia.

The very rare solid tumours are usually, according to Winckel, triangiomatic myxosarcomas. They do not show a malignant

character, as there was apparently in no case a recurrence, although at the extirpation radical removal until healthy tissue had been reached was by no means always undertaken (Stöckel). Herweg describes a case of myxangioma at the placental end of the umbilical cord in conjunction with stenosis and aneurysmal enlargement of the umbilical artery.

The other congenital changes in the umbilical stump are of less importance clinically. In addition to the varicosities already mentioned so-called false nodules may occur, and sometimes there is a remarkable development of Wharton's jelly which may delay the drying of the cord.

The various changes and abnormalities in position of the umbilical cord, which may threaten the life of the infant in utero, belong to the domain of obstetrics, and are only of interest to the pathology of the newborn in so far as they may be the causes of asphyxia. This group includes torsion of the cord, stenosis of its vessels from disease, compression of its vessels owing to prolapse, or the formation of nodules, or hæmatomata or winding of the cord around parts of the body. Injuries in cases of velamentous insertion of the cord, or laceration, may also threaten the infant owing to hæmorrhage.

GANGRENE OF THE UMBILICAL STUMP.

The umbilical stump may become gangrenous during the first week instead of drying up. It becomes discoloured and greasy, and the decomposing tissue secretes a brownish discharge which is usually very foul. The gangrene may be limited to the distal portion, so that when this separates a small shrivelled stump remains, from which the open end of the umbilical vessels is often seen projecting. In the course of a few days the remaining tissue falls off after a serous or sero-purulent discharge (Knöpfelmacher). This process may be accompanied by fever. The putrid stump is a good nutrient medium for the growth of bacteria, and the umbilical wound may become infected, but general septicæmia is a comparatively rare sequel.

Treatment consists of the immediate removal of the gangrenous portion. If any healthy cord remains at its insertion it should be clamped at this point with forceps, and cut off with scissors or knife immediately above. The cut surface must be washed with peroxide solution, cauterized with nitrate of silver, and thoroughly dusted with *dermasol*. The removal of the gangrenous portion by means of the thermo-cautery is a very useful method.

In order to avoid gangrene, it is important to see that evaporation of moisture from the cord is not prevented, for which reason it should be wrapped in absorbent gauze, and measures should be taken to hasten the process of desiccation. The daily bath has been blamed for the occurrence of moist gangrene, but, despite this, gangrene is not likely to occur if the dressing is aseptic and not too

thick and if a drying powder is used as previously recommended. The best method of prophylaxis is, however, not to leave a long stump, but to cut it off short.

(3) Diseases of the Umbilical Wound

DELAYED HEALING, AND MILD INFLAMMATORY AFFECTIONS
(MOIST UMBILICUS, EXCORIATION OF UMBILICUS, BLENNORRHOEA OF
UMBILICUS, CATARRHAL OMPHALITIS).

The slight disturbances which interfere with the healing of the umbilicus are very numerous, and Runge is quite justified in his statement that there is no well-defined line between a healthy and an unhealthy umbilical wound. The moisture exuding from it varies in amount; it may be serous or sero-purulent. This probably depends upon the amount of granulation tissue formation under the loosening portion of cord. The reaction which accompanies the separation of a very gelatinous cord is more intense than that which occurs with a less gelatinous cord. Further, if the cord is very thick, it is obvious that the umbilical wound will be large. In addition to the exudation there may be slight swelling and redness of the skin. This is partly due to the inflammation in the umbilical wound and partly due to the moistening effect of the discharge. Runge states that the healing process is normal as long as no increase of the redness, swelling or discharge occurs after the timely separation of the cord. "As the stump causes the umbilical inflammation, the latter must cease when the stump falls off; but if it increase, there must be some other cause for it."

It must also be remembered that many moist affections of the umbilical wound—which are usually harmless—originate in bacterial infection, the source of which may not be only in the cord, but in the adjacent skin, or even in some external cause. These affections frequently appear during the first week while the cord is separating. The gauze covering the umbilicus is moistened with discharge, and if the cord is lifted off the umbilicus with a probe, it will be seen that the umbilical surface is secreting more or less abundantly. After the cord has fallen off the umbilicus looks like a granulating wound. Even if the secretion which exudes therefrom contains numerous pus cells, the discharge does not usually look purulent. The much rarer cases in which definite pus is found in the folds and the recesses of the umbilical wound are termed *blenorrhoea* or *pyorrhoea* of the umbilicus.

To sum up, an intensification of the process of wound healing may occur, accompanied by complications probably due to bacterial agency. This causes a more or less profuse secretion of serous, sero-purulent, or more rarely, completely purulent exudate. The process is always local at first, and usually remains so, but it may produce a severe disease of the umbilicus, or it may form the starting point of a general infection.

SEVERE INFLAMMATORY DISEASES OF THE UMBILICUS
(ULCER OF THE UMBILICUS, OMPHALITIS, PHLEGMON AND GANGRENE).

The amount and virulence of the bacterial infection will determine whether any of the slight disturbances just described will be followed by more serious disorders of the umbilical region. The natural resisting power of the infant will obviously be an important factor in the matter.

If ulceration takes place in the region of the umbilical wound the affection is termed *ulcus umbilici*. In such cases the umbilical base forms a discoloured or greenish-grey tumour, which exudes serous or sero-purulent secretion, and is frequently covered with a fibrinous, firmly adherent coating. The skin surrounding the navel shows either no changes or it is swollen and red. Occasionally the folds surrounding the navel are stuck together with dried-up secretion, so that only after its removal is the ulceration visible. In rare cases the ulceration may also affect the surrounding skin. As Knöpfelmacher remarks, the walls of vessels offer more resistance to ulceration than the other tissue, so that sometimes the stumps of the umbilical arteries may protrude from the floor of the ulcer. The bacteria which cause the process of ulceration are, in the majority of cases, common pyogenic. In some cases the deposit on the ulcer was shown to be caused by the diphtheria bacillus (Hassenslein, Gentler, Toch, Finkelsiein); occasionally diphtheria antitoxin serum has been used with success. With such conditions it must be supposed that in superficial exudations avirulent diphtheroid bacilli, pseudo-diphtheria bacilli, &c., frequently collect; syphilitic umbilical ulceration has also been described. (See p. 515.)

The inflammatory disease of the umbilical wound may also attack the neighbouring skin. It is frequently irritated, macerated and eroded by secretion. Occasionally secondary infections develop in the form of small pustules which may develop into bullae; the term then used is *pemphigus neonat. periumbilicalis* (Trauteneck).

The term *omphalitis* is applied to that condition in which the inflammation spreads to the cellular tissue of the neighbouring skin. It is manifested by redness, infiltration and swelling of the skin surrounding the navel, sometimes in its close vicinity, sometimes in a large area of the abdominal skin (fig. 78). The inflamed part of the skin, infiltrated, stretched and shiny, is generally conically protruding. Towards the central summit of the protrusion, which is formed by the navel, and which may be suppurating, agglutinated by secretion, displaced by oedematous folds of skin, ulcerated or already cicatrized, enlarged veins converge, sometimes also lymphangitic streaks. Touching the child gives it extreme pain, forcing it into a certain position; the legs are drawn up towards the trunk, the type of breathing is mainly costal (Runge).

The condition is a dermatitis (allied to erysipelas) or a phlegmonous process. The disease generally occurs after detachment of the remainder of umbilical cord, in the second or third

work. The general symptoms (fever, prostration, loss of appetite, &c.) depend on the severity of the local inflammatory process and on the general septic complications, which, of course, may readily occur.

The local symptoms, in mild cases, may subside spontaneously. Sometimes, but not often, abscesses develop. The inflammatory process may affect the umbilical vessels and lead to arteritis and phlebitis. It may also spread internally and attack the abdominal walls and finally the peritoneum. The result is then fatal peritonitis.



FIG. 28.—Omphalitis and abscess of the umbilicus.

General infection may occur without disease of the umbilical vessels and peritonitis.

The most severe local disease of the navel is gangrene of the umbilicus. Before the antiseptic period the disease was frequent like hospital gangrene generally; nowadays it is only exceptionally observed and must be almost unknown among medical men of the younger generation. But that it is not merely of historical interest can be gathered by a statement from Hoché in 1902, who reported six fatal cases of umbilical gangrene, in the practice of one midwife in seven years.

The disease appears to be caused by the particular virulence of, possibly, specific organisms. It starts primarily from an ulcer of the umbilicus or omphalitis, or from severe local disease of the navel, in sickly, ill-nourished children, particularly during the course of septic conditions (secondary gangrene). The clinical symptoms of umbilical gangrene are described by Runge as follows: The border of an inflamed umbilical wound becomes discoloured and breaks down, giving rise to a more or less large loss of substance, or a vesicle is found, particularly in omphalitis, with opaque contents, which bursts and leaves an ulcer. The moist gangrene spreads rapidly, and may extend superficially or deeper. The gangrenous part is surrounded by a reactive inflammatory area; gradually gangrenous shreds are separated and a fetid smell issues from the diseased part. Fever is not obvious, as symptoms of collapse soon occur.

If the child is in a vigorous condition and the disease only spreads slightly, recovery may occur with separation of the gangrenous part. In other cases the process spreads further. If it goes deeper it affects the peritoneum. Diffuse purulent peritonitis follows or gangrene of the intestine which ruptures either into the peritoneal cavity, or, after adhesion to the abdominal wall, externally (Widerhofer, Plappart) (fig. 20). If the abdominal wall has a large perforation a whole loop of gut and its mesentery may protrude (Furth).

The duration of the disease varies; it may last a few days or several weeks. The prognosis is very bad, the mortality about 85 per cent.

Treatment of Inflammatory Umbilical Diseases.

The exuding and coated umbilical wound is best cleaned daily, once or twice with H_2O_2 (2 to 3 per cent.) or painted with 2 to 5 per cent. silver nitrate solution and then dusted with dermatol, xeroform, iodo, &c., or with bolus alba or, according to Runge's recommendation, with salicylic acid powder (acid salicyl. 1 : talc. 5) and then bandaged. If the umbilical cord is about to separate, but still adhering, care must be taken to apply the preparation beneath the stump.

In ulcerative processes the floor of the ulcer can be cauterized with a silver nitrate pencil or with the thermo-cautery.

In severe inflammatory processes in the region of the navel, antiphlogistic compresses with lead lotion are best applied. Abscesses must, of course, be incised, best on the dorsum.

In gangrene, one should attempt to confine the area by destruction of the tissue with the thermo-cautery right up to the healthy tissue. Runge recommends air-tight vapour compresses with subacetate of lead, which hastens the release of the slough. After this has been effected further treatment should be the same as with moist navel.

Thanks to the importance given to the aseptic care of the navel,

umbilical gangrene is a rarity, and the existence of other infective umbilical diseases can be avoided by aseptic umbilical treatment. Prophylaxis therefore takes the most important place in the treatment of umbilical diseases. As already mentioned, the difficult point of prophylactic care of the navel does not consist in the choice of a certain method of ligature and division, nor in a certain kind of umbilical bandage, nor in the question as to the daily bath, but in the strict carrying out of general aseptic rules. The best method is illusory if the after treatment is careless, e.g., if



FIG. 79.—Umbilical gangrene with perforation of the abdominal wall.

the umbilical bandage is displaced, so that various bacteria can easily enter the exposed umbilical wound. If real care is taken, that ligature and division take place under perfectly sterile conditions, that the navel is aseptically ligatured and the bandage is properly applied, if the wound is daily inspected and every minute disorder is

immediately attended to, such procedure is of far more importance than all ideas of improving on the various methods of umbilical treatment.

TUMOURS IN THE REGION OF THE NAVEL.

The most frequent and the most important form of tumour at the navel is the granuloma (*fungus umbilici*, sarcomphalus, fungating growth). The affection is simply due to the formation of granulations; these little tumours occur, therefore, after detachment of the cord, in the second or third week, and generally on a weeping umbilical wound. They take the form of tumours, either flesh, strawberry or raspberry coloured, sometimes barely the size



FIG. 80.—*Granuloma umbilici*.

of a pea, and sometimes that of a walnut, and are situated on underlying tissue, sometimes with a broad base and sometimes pedunculated (fig. 80). The granulations may, in exceptional cases, surround the stumps of the umbilical arteries, which then hang from the umbilical wound in the form of long, loose tags (Finkelstein). As the granuloma exudes much secretion, inflammatory and eczematous changes readily occur in the region of the neighbouring skin. The growth, if left to itself, may last for months and increase in circumference. When healing finally occurs, small nodules of skin or tags remain at the navel, such as may sometimes be observed in adults.

Granulomata can easily be removed. In small growths frequent

touching with the silver nitrate pencil is sufficient, and then application of drying powder. Large fungating or pedunculated granulomata are best ligatured with a sterile silk thread; they either fall off spontaneously or can be removed with curved scissors. The granulomata can also be removed with a small sharp spoon or by means of the thermo-cautery. If there is much bleeding, pressure with a silver nitrate pencil is generally sufficient to staunch it. Owing to the nervelessness of granulomata all the above procedures give no pain to the child.

Similar to granulomata, but quite different in their anatomical structure, are those small umbilical tumours which are termed adenoma (Küstner) or enterocœloma umbilici (Kolarczek). They consist of numerous gland ducts covered with columnar epithelium and of unstriated muscle, and are probably due to a prolapse of the ductus omphalo-mesentericus. They are therefore remains of an external part (generally a blind end) of Meckel's diverticulum. After excision of an umbilical adenoma, Lannibongus and Frémont observed a faecal fistula remaining; this points with all the more certainty to a connection with Meckel's diverticulum. Care must be taken not to confuse these fungating tumours of the umbilical region with a diverticulum or prolapse of the intestinal mucous membrane through an open diverticulum with prolapse of the wall; the discovery of the mouth of a fistula makes the diagnosis certain.

Fungoid adenomatous formations at the navel may possibly be due to the remains of the allantois.

Virchow describes a peculiar "omphaloid," hyperplastic, congenital formation at the navel resembling spindle cell sarcoma (of the same construction as the umbilical cord), which protruded from the umbilical ring like a very red coloured horn (Range).

(4) Diseases of the Umbilical Vessels.

The inflammatory diseases of the umbilical vessels are due either to infection of the inner coat of the vessels—thrombo-arteritis and phlebitis—or of the perivascular connective tissue and the lymphatic vessels of the adventitia—periarteritis and phlebitis.

(a) ARTERITIS UMBILICALIS.

Thrombo-arteritis occurs in this manner: from an infected navel or blennorrhœa or an ulcer the inflammation spreads to the thin thrombus of the umbilical arteries, and the latter breaks down. As a rule the suppuration extends only to the peripheral parts of the arteries. There is then copious exudation of pus from the umbilicus. If the abdominal region is stroked in the direction of the vessels towards the navel the pus sometimes comes out of the vessels still more copiously. By means of a thin probe one can sometimes pass right into the arteries. Otherwise the clinical aspect of the disease resembles local pyorrhœa of the navel, and is only distinguished from it by the spreading of the suppuration to the thrombi in the terminal parts of the arteries.

Thrombo-arteritis extends further into the vessels only in rare

cases; but just occasionally the purulent process may spread to the origin of the umbilical arteries from the *A. hypogastrica*—thrombo-arteritis totalis (Escherich, Finkelschein). The danger of a general infection is very considerable in such cases.

Far more frequent than thrombo-arteritis is periarteritis umbilicalis. Of all the dangerous umbilical affections Runge considers it the most frequent, and therefore the most important. The pyogenic organisms may easily pass along the perivascular tissue of the arteries and their lymphatics; this may occur more easily than in the lumen of the arteries, the ends of which are much contracted, and more easily than in the perivascular tissue of the umbilical vein, as, according to Runge, the connective tissue ring surrounding the transverse section of the umbilical artery is about twice as thick as that of the vein and adheres much closer to the navel than the latter; therefore the passage to the arteries is broader and more accessible for bacteria.

Periarteritis always attacks both vessels, either in the distal parts only or throughout their whole extent. It is, clinically, remarkable that the inflammatory signs occasionally begin at the distance of 1 to 2 cm. from the navel. The inflammatory or phlegmonous disease of the arterial coverings leads to considerable thickening and rigidity of the wall of the vessels. The inflammation may spread secondarily from the perivascular tissue to the wall of the vessel itself, and finally to the interior of the vessel; in the lumen also are found collections of pus which dilate the vessel as a whole or only in parts.

In other cases the process is almost exclusively perivascular, and the lumina of the vessels are very narrow.

The infection may occur while the cord is still attached, or after it has fallen off. The time when the infective organisms gain access depends entirely upon circumstances; if the liquor amnii is infective, intra-uterine infection may take place. As a rule, it occurs by contamination of the cord with infective materials, e.g., lochial discharges, through cutting the cord with an unclean instrument, or enveloping the cord in a dirty dressing, or touching it with dirty fingers, or by means of dirty bath-water or sponges. Bacterial action may produce local changes in the cord or the umbilical wound, but the action may be exerted in the deeper tissues, so that umbilical arteritis may be secondary to a local umbilical disease, or it may be a primary condition, the cord itself appearing to be quite healthy.

Streptococci and staphylococci are the most important organisms concerned; infection by diplococci and *B. coli* is rare (Porak and Durante). Wassermann describes an epidemic of umbilical arteritis caused by *B. pyocyaneus* in which eleven infants died.

The symptoms and course of the disease vary widely in individual cases, and depend upon the extension of the process, the virulence of the organisms and resistance of the infant. The disease is most serious in premature infants.

In many cases the course of the disease is quite favourable;

indeed, cases frequently run their course to recovery without being recognized. According to the researches of Finkelstein and Escherich, encapsulated abscesses can become absorbed, leaving only a scar. But it is not only the favourable cases which may present no symptoms; for, as Runge suggests, death may result from unsuspected umbilical arteritis. An apparently healthy infant may suddenly become restless, refuses nourishment and dies—the post-mortem shows umbilical arteritis. An infant with apparently a simple local umbilical disorder may also die quite suddenly—obviously there must be an acute toxæmia in such cases.

Suppurative processes in the umbilical arteries may give rise to septic conditions which are at once recognizable clinically. In some cases pyæmia will develop with metastatic lesions in various organs, especially the lungs and joints. In all these cases there may be a complete absence of local signs, or they may be very slight. Changes may be visible in the cord, or it may look quite healthy. If the arteritis and its sequelæ develop slowly, the umbilical wound may have healed over entirely.

Pur only exudes if the peripheral portions of the arteries are diseased, and this symptom is not of frequent occurrence; but the umbilical hæmorrhage which occasionally occurs must be regarded as a sign of sepsis. If the area is carefully examined with a probe a sinus will sometimes be detected, which leads into a perivascular channel of varying length, which has developed in the decomposing tissue. Sometimes the thickened arteries can be felt as cords through the abdominal wall beneath the umbilicus. In these cases it is usual for fairly large abscesses to develop, and they are frequently followed by sequelæ which depend upon an extension of the suppurative process. If the suppuration extends superficially a pre-peritoneal phlegmon will form, and this may travel along the inguinal canal into the scrotum, or it may reach the thigh (Finkelstein). If the suppuration extends deeply, purulent peritonitis may develop eventually (K. Mayer).

Umbilical arteritis may form the basis of a chronic sepiæmia which manifests itself clinically in various forms of infantile atrophy.

An infant, aged 5 weeks, was admitted to the Children's Hospital, Vienna, owing to its failure to obtain adequate nutrition from breast-feeding. The infant looked pale, its skin was flaccid, and persisted in losing weight although it apparently took enough from the breast. There were no obvious signs of disease, and the temperature was not raised. The case was designated "atrophy on breast-feeding." The infant died in ten days with symptoms of pulmonary infiltration. Post-mortem. Two abscesses containing empysematous pus found in the umbilical arteries beneath a completely healed and apparently normal umbilicus. There were abscesses in the left liver lobe and a fibrino-purulent pleurisy.

The duration of the disease is very indefinite, and the clinical symptoms afford little help in determining it. It depends mainly upon complications. It is difficult to fix the beginning of the disease. Runge observed five cases where a death was definitely due to umbilical arteritis; the oldest was eighteen days old and the youngest four days old. The greatest mortality occurred

on the eighth day. The conclusion follows that infection occurs, in most cases, within the first week, while the cord is still attached.

(6) UMBILICAL PHLEBITIS.

For anatomical reasons already explained, inflammation of the umbilical veins occurs much less frequently than that of the arteries. In some cases, however, the veins and arteries are both affected.

Whereas arteritis not infrequently ends in recovery, despite its dangers, phlebitis is invariably fatal. The disease affects the entire vein as far as the liver and often causes disease of the hepatic vessels, of Glisson's capsule, or of the liver itself (abscesses, diffuse hepatitis). But the condition is dangerous, apart from any hepatic complication. As soon as the intima of the vessel is involved, the micro-organisms are carried to the heart and the general circulation.

In phlebitis, the absence of local symptoms in the umbilicus is more frequent than in arteritis. It is never possible to extract pus by massage, but blood sometimes trickles out of the umbilicus.

The clinical symptoms are those of septicæmia. Jaundice is one unfailing characteristic of umbilical phlebitis, and this may become so intense as to produce a brown discoloration of the skin (*ictère bronze*—Ponak and Durau).—The infants have a raised temperature, as a rule, and are very restless. Widerhofer has drawn attention to a special type of respiration which is often associated with umbilical phlebitis: the inspiration is short, the expiration is prolonged, and the respiratory rate is increased, the thoracic movements are hardly perceptible, and the abdominal movements are entirely arrested. The cause of this peculiar breathing is probably the pain produced by the peri-vascular inflammation in the upper abdominal region. For the same reason, the lower extremities are drawn up towards the abdomen, as in omphalitis. But these symptoms hardly suffice to establish the diagnosis with certainty.

The precautions described in connection with the prophylaxis of local diseases of the umbilicus also apply to the prevention of disease of the umbilical vessels. Local treatment is also essentially the same. If there be any communication between the inflamed area and the umbilicus itself, it is important to remove by means of a probe any obstruction caused by discharge or folds of the mucous membrane at the external orifice. The latter should be carefully spread out so as to render it possible to irrigate the inflamed focus with a disinfectant solution.

An infant was suffering from a severe general infection with a high temperature for nine days. The discharge from an arterial thrombotic abscess was evacuated by means of a probe, and the symptoms disappeared suddenly and permanently.

The therapeutic measures already detailed are to be employed for cases of general infection resulting from inflammation of the umbilical vessels.

Hæmorrhages from the umbilical vessels will be referred to later on (v. p. 436).

PART VII

Hæmorrhages and Hæmorrhagic Diseases of the Newborn Infant

The newborn infant often suffers from ruptures of the smaller vessels and effusion of blood into the tissues. These vascular lesions are only rarely the direct result of injury at birth, and most of the hæmorrhages found in the skin, mucous membranes or viscera are due to congestion. There is a certain amount of venous congestion attendant on every birth. The congestion may be local and confined to the region of the caput succedaneum, or it may be general. The longer the duration of the period of expulsion, and the disturbance of the circulation and respiration which accompanies it, the more is the infant liable to congestive processes. It follows, therefore, that congestion occurs most frequently in infants who are born in a condition of asphyxia (asphyxial hæmorrhages or ecchymoses). In most cases the hæmorrhages which arise from this cause are quite harmless; they consist of punctiform ecchymoses such as often follow an attack of whooping-cough. When the congestion ceases the hæmorrhage stops, the vascular lesion heals up and the effused blood becomes absorbed.

The sequelæ of these birth hæmorrhages depend upon the size of the injured vessels, upon the degree and duration of the accompanying congestion and upon the localization. It is indeed only in cases of hæmorrhage within the cranial or vertebral canal that serious consequences may ensue for the infant. As previously mentioned, these cases often become serious because they are associated with persistent congestive processes which give rise to considerable hæmorrhage from the injured vessels. Incidentally, a word of warning may be uttered against careless methods of resuscitation. The congestive ecchymoses are mostly capillary, and therefore, even if the congestion is long persistent, the amount of blood effused is slight.

The danger of birth hæmorrhages—including traumatic and congestive hæmorrhages—consists of the mechanical effects of the effusion, *viz.*, the destruction of tissue and the pressure. The actual amount of blood lost is of secondary importance. The pathological results occur during birth and the first few hours afterwards. As a rule the immediate danger of a birth hæmorrhage ends with the first day, although sometimes its result appears later. In some cases, however, careless movements may cause a return of hæmorrhage from one of the larger vessels after it has once ceased.

In addition to these hæmorrhages, there is another type which affects infants in the very early days of life. This is analogous to the condition which is termed "hæmorrhagic diathesis" when it occurs in older infants. Among the latter the hæmorrhages appear in the form of purpura, subcutaneous and mucous membrane hæmorrhages, whereas in very young infants the bleeding occurs in the digestive tract. This tendency of the newborn infant to bleed from the intestinal canal is expressed by the old term of *melæna neonatorum*, which signifies diseases accompanied by the vomiting of blood and by blood-stained stools. The digestive canal is, however, not the only seat of hæmorrhages even in the newborn infant. Various other tissues and organs may be involved, such as the adrenal glands, the liver, the lungs, the mucous membrane of the mouth and nose, and the umbilical wound. Hæmorrhages occur but rarely from the skin and subcutaneous tissue, at any rate not those patchy and punctiform hæmorrhages which are characteristic of purpura. In all hæmorrhagic diseases of the newborn the loss of blood is very profuse and the consequent anæmia is severe. Their period of onset is always within the first ten days of life. The cause of these diseases is by no means clear—a point we shall have to refer to later on. It is doubtful whether they have a uniform causation; there may be several poisons which act as exciting causes for the hæmorrhages. Observation teaches us that the clinical picture as seen in the newborn infant hardly ever occurs in later infancy in an analogous manner. But the hæmorrhagic diathesis of older infants, already referred to, evokes similar symptoms. In these cases, however, we are concerned with a definite "diathesis," with a predisposition to disease, in Pfäundler's sense, in virtue of which the same infant is subject to repeated attacks of the disease; but in newborn infants the predisposition to hæmorrhagic disease depends upon the age period and not upon the individual. If an infant has had an attack of *melæna neonatorum*, it is not followed by a tendency to hæmorrhagic diseases in later life. The tendency to hæmorrhages ceases as soon as the period of early infancy has passed, showing that they depend upon some physiological peculiarity of this age-period. This consideration justifies us in discussing these diseases from one common standpoint, as American writers have done under the designation of "Hæmorrhagic diseases of the newborn"—excluding, of course, birth injuries and severe secondary hæmorrhages (Holt, Greef, Lambert, Swain, Jacobson, Murphy, Schloss and Commiskey, Moss and Gillen, Macneil, MacClanahan, Abt, Tukey, Kilham and Mercalis, Grünberg, Graham, Young and Richards).

Hæmorrhage from the Gastro-intestinal Canal.

MELÆNA NEONATORUM.

The term *melæna* (black disease) only means the excretion of blood from the bowel. It is merely a symptom, and as a matter of

fact a number of heterogeneous diseases are included in the term *melena*.

It is the custom to distinguish *melena vera* from *melena spuria*, the latter including all cases of excretion of blood wherein the blood did not originate in the gastro-intestinal canal, but had found its way therein by swallowing. The swallowed blood may come from the mother or from the infant. In the former case its source is either some on the nipple or the maternal genital tract. Blood may easily get into the mouth of the infant during its passage through the vagina, if the mucous membrane is injured, or if a varix bursts, or from a perineal laceration or after episiotomy. The infant will then frequently vomit a brown-coloured fluid, often mixed with mucus, during the first twenty-four hours. This symptom is quite harmless. The meconium may show traces of blood on chemical examination on the first day, although the general appearance of the stools is not typical of *melena*, the amount of blood swallowed being too small to cause this appearance. Larger quantities of blood may be swallowed if there has been early hæmorrhage owing to the situation of the placenta (Baisch), or if a vessel has been torn in a placenta with a velamentous insertion. Certain cases of *melena* of intra-uterine origin may be explained in this way (Schlicke, Kamann), being caused by intra-uterine movements of deglutition or premature attempts at breathing.

The swallowed blood may come from the infant in cases of injury to the base of the skull (Reinhold, Hodges), to the mouth cavity or to the pharynx, or in hæmorrhages of the naso-pharyngeal space. The latter are of the greatest clinical importance, because there may be a perfect resemblance to a genuine *melena*, especially if there be no external bleeding from the nose. It is therefore necessary to examine the posterior pharyngeal wall to see whether any blood is trickling from the roof of the pharynx. The examination must be conducted carefully with good illumination, because it is very difficult to obtain an adequate view of the posterior pharyngeal wall. Strictly speaking, nasal spurious *melena* is the only form which properly belongs to the class of hæmorrhagic diseases. Even if the hæmorrhage does not proceed from the gastro-intestinal tract, it still may be as extensive in amount and cause as great a degree of anemia as in cases of severe *melena* (Svoboda, Hochsinger, Lalmer). The other forms of spurious *melena* ought to present no diagnostic difficulties, if the infant and the mother's breast are carefully examined and if the contingent circumstances are taken into consideration. As a rule the amount of blood lost is considerably less than in cases of genuine *melena*.

Melena vera comprises those hæmorrhages wherein the source of the blood is also in the gastro-intestinal tract; but among these there are some which may be regarded as symptomatic *melena*, the hæmorrhage occurring in the course of some general disease which is manifest clinically. The various forms of septic disease are the most important in this connection. But nevertheless the course of

septic disease is not usually attended by profuse hæmorrhages, the vomiting of blood and the excretion of dark red stools. Conditions very similar to those of hæmorrhagic sepsis may occur in syphilitic children; but nevertheless the typical picture of melæna is of very rare occurrence in syphilis. The forms of melæna which are associated with enteritis must also be regarded as symptomatic. In these cases the intestinal hæmorrhage is preceded by prodromal signs: persistent vomiting without blood, diarrhœa, colic, abdominal distension and frequently a rise in temperature (Shukowski.) The condition is either one of hæmorrhagic enteritis (gastro-entero-colitis hæmorrhagica) or hyperæmia of the intestinal mucous membrane with permeability of the vessels, or it may be a septic infection arising in the bowel. In these circumstances the hæmorrhage is associated with symptoms which obviously point to inflammatory processes in the mucous membrane of the digestive canal. In most of these cases, however, there is no profuse hæmorrhage as in a typical melæna, but merely an admixture of blood with the stool.

In a narrower sense, *melæna vera* expresses the condition wherein the gastro-intestinal hæmorrhage dominates the clinical picture, in the absence of any evident symptoms of any other general or local disease. In this limited sense of the term, melæna does not represent a disease of uniform ætiology. From the standpoint of pathological anatomy we may advance a step further and describe an idiopathic melæna within the group of *melæna vera*, indicating by this term a type which reveals no cause whatsoever for the hæmorrhage on post-mortem examination. *Melæna vera*, however, must be allowed to remain as a clinical entity for the present.

Melæna vera usually begins between the second and fifth day of life, more rarely on the first day or towards the end of the first week. Typical "idiopathic" cases hardly occur at all after the tenth day. Vassmer's statistics show that the disease begins, in most cases, on the second day. The hæmorrhages which start after the fifth day are generally definitely symptomatic. The blood may either be vomited or passed with the stools. Hæmatemesis by itself is rare; even if the vomiting of blood is the prominent symptom, enough blood remains in the bowel to ensure its excretion with the stools after a little while. In other cases, hæmorrhages from the stomach and from the bowel occur together; but very frequently the blood only comes from the bowel.

Vassmer gives the following table:—

| Blood | Commencement | Duration | Mortality |
|-------------------------|--|--|----------------|
| 6 cases only in vomit | 2 cases, 1st day 2 " 2nd " | — | 85·5 per cent. |
| 20 cases only in stools | 1 case intra-partum 5 cases, 1st day 9 " 2nd " 3 " 3rd " 1 case 4th " 1 " 5th " | 7 cases 1 day 1 case 1½ day 2 cases 2 days 3 " 4 " " " " | 10 per cent. |

| Blood | Commencement | Duration | Mortality |
|---------------------------------|-----------------|---------------------|----------------|
| 32 cases in vomit and in stools | 8 cases 1st day | ... 1 case 11 hours | 57·1 per cent. |
| | 26 " 2nd " | ... 2 cases 1 day | |
| | 7 " 3rd " | ... 12 " 2 days | |
| | 1 case 4th " | ... 5 " 3 " | |
| | 1 " 5th " | ... 1 " 4 " | |
| | 1 " 10th " | ... 1 case 5 " | |
| | | 8 cases | |

The typical melæna stool appears during the meconium stage or in the transitional period. The hæmorrhage starts before the ordinary milk stools have appeared. No chemical test is required to detect the blood in typical cases; it is at once noticeable that the motion is not of the blackish green or brown colour of the meconium. The melæna stool is blackish brown and usually streaked with red, it is much more bulky than meconium, and is of less tough consistence. The admixture of blood often betrays itself by a definite blood-red area in the napkin, which surrounds the brownish-black faeces. It usually has the very characteristic odour of decomposed blood. At any rate, it is not odourless like meconium, nor has it the acid smell of the transition period or of the milk period. The stools retain the same characteristics, whether the child takes nourishment or not. In bad cases the stools increase in frequency, and become more and more bloody, containing also coagulated blood. If the coagulability of the blood is defective, the stools consist of thin pasty or almost liquid dark red masses; in other cases they are more coherent and of the consistence of coagulated blood. The blood may trickle from the anus in a thin liquid form, so that the napkins are always saturated, however frequently they may be changed.

As recovery sets in, the cessation of bleeding is indicated by the gradual diminution of the red colouration in the stools. The bowels act less frequently, and the faeces become of a smoky brown colour, owing to the longer stay of the blood pigment in the intestine and the consequent chemical change which it undergoes. Sometimes the hæmorrhage ceases quite suddenly. After a very definite melæna stool a yellowish-brown action may appear, containing remains of milk, in which no blood can be detected even by chemical methods.

The disease is ushered in occasionally by the vomiting of blood; but this symptom does not, as a rule, remain the only one, unless the case is so grave as to lead to a rapid death. In most cases blood appears in the stools within a day or two. The vomiting of blood ceases after a couple of days; in rare instances it may persist long and dominate the clinical picture. The vomit consists of pure liquid bright red blood, mixed with clot and mucus. Sometimes the vomit is brown, similar to the vomit of maternal blood which has been swallowed by the infant, as previously mentioned.

In most cases the hæmorrhage is limited to the digestive canal, but it may occur occasionally in other situations, e.g., the mucous

membrane of the mouth, the palate, the nose, and the internal organs. Deep subcutaneous hemorrhages are noted sometimes.

If the disease be fatal, the anæmia, which develops rapidly after the profuse losses of blood, may become very profound. The skin looks like wax and is sometimes as white as paper. The visible mucous membranes appear to be quite devoid of blood. The colour of the conjunctiva, the lips and the mucous membrane of the mouth hardly differs from that of the adjacent skin. There is a continuous and gradual loss of blood, which eventually becomes quite thin, and finally quite watery, flowing from the mouth, nose and rectum until death occurs.

A severe anæmia may develop even in those cases which end in recovery, because the loss of blood may be profuse, although the duration may be brief. More or less pronounced jaundice may occur, but it may be nothing more than a slight yellowness of the skin in cases of gastrointestinal hæmorrhage. In some cases there is no jaundice at all, obviously because the material which forms bile pigment has been poured out into the intestinal canal and has been evacuated.

The temperature is usually raised, being sub-febrile or febrile; but the pyrexia is transitory. It is difficult to decide whether the fever is an expression of an existing morbid process or whether it has any connection with the melæna, with which it is coincident in point of time. Sometimes the temperature remains quite normal, even in vigorous infants, so that it is impossible to say that constitutional weakness or the amount of blood lost is responsible for the temperature not rising. After severe hæmorrhage the temperature may become sub-normal.

At the beginning of the disease the taking of nourishment is hardly disturbed, and if the disease remains mild or moderately severe there is no interference with the feeding, but if the loss of blood is great, the infant becomes drowsy and ceases to suck. The decrease in weight is greater than that which should correspond to the physiological loss. The decrease in weight is also very considerable in the cases which eventually recover, and the infant only puts on weight very slowly. The duration of the disease as well as its severity is very variable. In some the hæmorrhage is so great that death is inevitable, and these are transitional forms down to those which are characterized by a few blood-stained stools, wherein the hæmorrhage is never alarming. The passage of blood does not last more than two or three days in moderately severe cases, and hardly ever persists more than five days. The severity of the case depends upon the amount and frequency of the hæmorrhage rather than upon its duration. If the evacuated blood is thin and very fluid, the case is serious because it indicates a lack of coagulability. If the hæmorrhage is confined to the bowel, the prognosis is better than in the cases where it is associated with hæmatæmesis. The average mortality, according to the accompanying table, is 50 per

cent., but the cases probably are not limited to *melena vera* in the strict sense of the term.

Mortality Tables.

| | | | |
|------------------------|--------------|-------------------|--------------|
| Dusser and Orie | 35 per cent. | Townsend | 70 per cent. |
| Mitte | 84 " | Silbersaum | 50 " |
| Klag | 35 " | Vander | 50 " |
| Ellis | 47 " | Sladkowski | 62 " |
| Tarrier | 10 " | Vassmer | 42 1/2 " |

The prognosis of the cases of idiopathic *melena* appears to be more favourable than that of other types of the disease. L. Unger lost only one case out of nine, and although three of the cases began with the vomiting of blood, the mortality rate was lower than in Vassmer's twenty cases of intestinal hæmorrhage only. It is quite certain that correct treatment reduces the mortality. If the infant survives, the prognosis is quite good because no sequelæ are to be apprehended.

THE ETIOLOGY OF *MELÆNA VERA*.

As the post-mortem findings are very varied, very different explanations of the disease have been offered. As sometimes nothing is found post mortem, numerous hypotheses have been advanced in explanation. There is no doubt, however, that the condition which we designate as *melena vera* does not possess a uniform ætiology, because the pathological and anatomical findings differ so much.

In attempting to arrive at the cause of *melena*, the following questions must be answered:—

(1) What is the nature of the vascular lesions which cause the bleeding?

(2) What is the cause of these vascular lesions?

(3) What is the cause of the persistence of the bleeding?

In order to answer the first, and to some extent the second question, it is necessary to dwell in some detail upon the pathological changes.

In some cases ulcers are found in the alimentary canal, viz., in the œsophagus, stomach, duodenum, and occasionally also lower down in the bowel. These ulcers are either single or multiple. von Preuschen found a single ulcer in sixteen cases and multiple ulcers in six cases; in three cases there were two to three ulcers. In twenty-four autopsies Dusser found ulcers in thirteen cases—in nine, ulcer of the stomach, and in four, ulcer of the duodenum. Vassmer collected twenty-two cases from the literature, and there were ulcers in twelve of these—three in the œsophagus, four in the stomach, five in the duodenum, and one in the ileum. The ulcers in the stomach are not usually deep (Binz); they are, as a rule, superficial peptic erosions, but the duodenal ulcers which are found more frequently penetrate more deeply, as far as the muscular layer or even to the serous coat. The œsophageal ulcers are the rarest.

Hemach and Spiegelberg have described vascular ulcers just above the cardia, and Karl Meier has observed a small ulcer in the lowest segment of the oesophagus.

But ulcers are not always found at the post-mortem in cases of melena. Baisch only saw them once in fourteen cases, and Heib states that they only occur in a relatively small number of cases. Shukowski, who has himself not had a case with ulceration, has shown from the literature that ulcers occur in about 45 per cent.; but this figure is probably too high because cases with ulceration are apt to be included in statistics, and the numerous cases where nothing is found are apt to be neglected.

Probably in most cases the anatomical findings are negative or very meagre. There is often a certain amount of hyperæmia of the mucous membrane, and sometimes there is injection in limited areas. Sometimes there are punctiform hæmorrhages, blood effusions of varying amount, and hæmorrhagic erosions in the mucosa. In one case Vorpahl found great venous dilatation in the middle third of the oesophagus, and he attributes the hæmorrhage to the rupture of veins in consequence of the contraction of the oesophagus during the act of deglutition.

If there be no lesion in the mucous membrane, one must assume that the hæmorrhage is parenchymatous—an exudation of blood corpuscles through the walls of the vessels and capillaries and through the apparently intact epithelium of the mucous membrane. Sometimes the microscope reveals round-cell proliferations in the mucosa (Schoeppler). Even if we find erosions and ulcers we cannot be sure that they are the only sources of the blood and that they explain the profuse hæmorrhage, because large vessels are not eroded as a rule; and, on the other hand, similar ulceration may exist in older infants without causing any hæmorrhage at all, or at any rate very little, e.g., the duodenal ulcers of atrophic infants. Further, numerous hæmorrhagic erosions are occasionally found in the gastric and intestinal mucous membrane of newborn infants which have caused no melæna during life.

Attempts have been made to correlate the hyperæmia, hæmorrhage and ulceration found in these cases with the process of parturition.

All infants are born with their internal organs to a more or less severe degree of congestion, and this is probably a contributory cause for the melæna. If the infant has a congenital abnormality of the heart, secondary congestion will follow and this leads to the production of gastro-intestinal bleeding, and the association of melæna with heart disease has indeed been noted frequently (Stannouds, Herrgott, Næberberg, Bauer).

The main cause of melæna has been sought for in the asphyxia and the venous congestion associated with it, for this condition can undoubtedly cause hæmorrhages. Landou suggests that there is an insufficient aspiration of blood from the umbilical vein in asphyxiated infants, so that thrombi develop in this vein or in the ductus arteriosus. These thrombi may produce embolic infarcts

and ulceration in the branches of the descending aorta and particularly in the duodenal arteries. Franquè has suggested a simpler explanation than this rather complicated one. He thinks that the clot which may develop in the umbilical vein may cause a retrograde embolus in the portal vein or its tributaries, and that conditions in the newborn infant are favourable to this process. Such a clot does not often develop, and therefore melena is rare. Franquè does not think that the asphyxia causes the formation of thrombi in the venous trunks, but he holds that the severe spasmodic respiratory and crying movements and the consequent variations in pressure are responsible.

In contrast to this view, which sees the cause of the development of ulceration in embolic processes, Rundstedt has endeavoured to explain it on the theory of "hyperemic hæmorrhages." He considers that softening is produced by hæmorrhagic infarcts, and that the gastric juice erodes some of these softened areas. In favour of the view that the ulceration is of the nature of a peptic ulcer is the fact that the ulceration is almost always found in the stomach and duodenum. Rundstedt argues, on the other hand, that hyperæmia does not necessarily cause hæmorrhage in the free lumen of the bowel, and that hyperæmia and hæmorrhage in the mucous membrane do not injure its vitality or power of resistance, as long as the epithelium remains intact. Benke offers the following explanation: Numerous asphyxial ecchymoses or typical necroses are found in the newborn without any indication of hæmorrhage. These are not caused by hyperæmia but by ischæmia. Benke, Rundstedt and Zudek think that this ischæmia originates reflexly in the nerve centres owing to the shock of birth (thermal or mechanical irritation, vascular spasm, &c.). This ischæmia is an indispensable medium in the series of processes which are required to produce necrosis of the epithelium, the erosion by the gastric juice, and hæmorrhage in the free lumen of the stomach or bowel.

The theory of a reflex nervous process is reminiscent of the older attempts at explanation by Preuschen and Pomorski. According to them, the ulceration in the gastro-intestinal mucous membrane is due to the formation of multiple effusions which are caused by lesions of the brain, especially in the vasomotor centre, and a consequent vascular atony with congestions and hæmorrhages. At first sight this explanation looks complicated, but it has been demonstrated by experiments on animals.

This theory definitely assumes that there is some connection between melena and injury at birth. As a matter of fact, melena does occur more frequently after prolonged and difficult labours. It is obvious that injury at birth merely acts as a predisposing factor, otherwise melena would be a much commoner disease. Most infants who are born asphyxiated and even with cerebral lesions do not develop melena.

An objection to the view that injury at birth is an antecedent of melena is its rare occurrence as an intra-uterine disease. In these

cases the autopsy reveals the presence of ulcers whose intra-uterine origin is proved not only by clinical signs, but by the presence of blood pigment on the floor of the ulcers (Dietel, Bährke, Wolfsohn, Holschmidt).

Schauz also attributes the lesions of the mucous membrane to damage by hæmorrhage; but he does not consider that the hæmorrhage is congestive, but rather a pathological exaggeration of a catarrh of all the mucous membranes which is physiological in the newborn infant. It is doubtful whether one is justified in speaking of such a "physiological catarrh" of the mucous membranes, especially such a condition in the intestinal mucous membrane. The question of catarrh depends upon the period when the melæna is most frequently observed. Schœppfer also assumes that melæna has some connection with this irritative catarrh. He thinks that the tying of the cord and the consequent interruption of the current of blood through the umbilical arteries may lead to acute hyperæmia of the gastro-intestinal canal, and that these circulatory disturbances suffice to cause hæmorrhages, especially if the feeding causes any further irritation of the intestine which is already damaged by the hyperæmia.

From the foregoing it is clear that vascular lesions are found in cases of melæna, in the form of laceration of vessels due to erosion or ulceration; but in many cases the post-mortem examination reveals nothing at all, or such trifling lesions that it is impossible to attribute the severe hæmorrhage to them. None of the suggested explanations enable us to answer satisfactorily all the questions which have been raised in regard to the nature and cause of the vascular lesion. All attempts at mechanical explanation fail in regard to those cases where the pathological findings are negative or insignificant.

If the vessel wall has suffered no injury, we must ascribe its permeability to *infective* or *toxic* damage. We know, from the pathology of later life, that general infections often cause hæmorrhages in the skin, in the mucous membranes, and various internal organs. It was therefore to be assumed that the profuse hæmorrhages in the digestive tract of the newly born were septic hæmorrhages, melæna being included as a hæmorrhagic form of sepsis.

The opinion, that melæna and hæmorrhagic diseases of the newly born are entirely due to septic origin, dates from a period in which septic hæmorrhages assumed such a dominating position that those forms to-day termed "idiopathic" retired, in contrast, entirely into the background. One may judge of the frequency of these diseases accompanied by hæmorrhages, when one reads in Epiton's report, that in one year, in the Prague Foundling Hospital, he observed 61 cases of hæmorrhage among 702 children!

The most important ground for the assumption of infection is the presence of bacteria in the blood. When the examination is positive it is, of course, possible to find objections to the ætiological significance of such bacteria. If the bacteriological examination of

blood is not undertaken till after death, it is not possible to exclude a post-mortem passage of bacteria into the blood, and all the more so if much time has elapsed after death before the examination is made. But even if the presence of bacteria is found immediately after death or during life, it may be still supposed that the sepsis only appeared after melæna. For the possibility of secondary sepsis arising is certainly indicated, owing to the deficiency of the wall between the blood and the interior of the intestine, such as may justifiably be assumed with melæna.

The results of bacterial examinations vary considerably. Practically all pathogenic micro-organisms, such as are found with sepsis, have not only been isolated from the blood, but have also been proved to exist in the intestinal wall and in the internal organs of children that died of melæna; streptococci, staphylococci and pneumococci, *B. coli*, *B. typhosus*, *B. enteritidis*, *B. lactis aerogenes*, *B. pyocyaneus*, *B. paratyphosus*, *B. haemorrhagicus*, and many others (v. Dungern, Baginsky, Bar, Neumann, Finkelstein, Orlowski, Tavis, Kilham and Mercalis, Nicholson, Rädler, &c.). Mixed infections have also been described. Gartner himself found, in two cases of melæna, what he regarded as a specific bacillus, belonging to the *coli-typhoid* group. Even if this Gartner melæna bacillus has not been found later, it has often been possible to isolate micro-organisms, which in experiments on animals caused hæmorrhagic inflammation (Finkelstein). Not much can be concluded from this, as changes in the vessel wall with accompanying extravasations of blood may be caused by various bacterial poisons.

The portal of infection for sepsis must, of course, be taken into consideration. The infection may come from the placental circulation, and therefore be intra-uterine. This passage of infection has incontestably been proved in a case of Nauwerck and Flinzer. From the blood of the body of a child that died on its second day from symptoms of melæna, these authorities were able to isolate a virulent strain of *B. paratyphosus* B morphologically, culturally, and by agglutination, which was agglutinated by the blood of the mother, who, a few weeks before delivery, had suffered with violent diarrhoea. Post partum the infection results less frequently from the remains of the umbilical cord than from the intestine. The opportunity for enterogenous sepsis is, in the newly born, particularly favourable, as the wall of intestine appears to be fairly permeable (Czerny and Moser). Czerny and Keller therefore attribute melæna neonatorum to enteral infections. They support their theory by the presence of fever and clanges of blood. Whether the condition of glycogenous degeneration of the leucocytes and many blood platelets is constant with all cases of melæna further research must show. In a case of Unger the leucocytes showed no iodophil reaction. As already mentioned fever is not a constant symptom of melæna, and if present is no proof of its septic aetiology.

It may be safely assumed that there is in reality a melæna of infective basis, the injury to vessels in some cases being attributable

to bacterial poisons. The variety of micro-organisms found does not contradict this theory. But that infection is at the bottom of all melena cases can hardly be assumed. The clinical course, the relatively early appearance, the sudden onset and disappearance of the symptoms, do not often correspond to the course of a septic disease. At the time when umbilical infections and also most enteric infections were first manifested, melena in its true typical form only exceptionally occurred. And, finally, the positive bacteriological findings stand in contrast to the negative (Schmorl, Baginsky, Unger, Schloss and Commiskey).

In those cases of melena in which there is neither demonstrable anatomical injury to the vessels, nor can permeability of the wall of the vessels from bacterial effects of poison be assumed, there must be poisons of a different nature that produce this injurious effect. We will deal later with this point, also with the third question as to the cause of the permanency of haemorrhages.

SUPRARENAL HÆMORRHAGE.

The soft, friable tissue of the suprarenal capsule, rich in venous vessels, appears to be a favourable soil for hæmorrhages. Congestive conditions and hyperæmia are found in the majority of suprarenal capsules of newborn children. The innermost cortical layers sometimes show a considerable capillary plethora (Kern). Mattei noticed the absence of hyperæmia of the suprarenal capsules only twice in thirty-nine cases; in five cases there were simultaneously extravasations of blood. Microscopically demonstrable hæmorrhages in the suprarenal capsule appear to be a constant condition in the newly born (Mattei, Philipp, Magnus). The larger effusions take place in the medullary substance, which may be entirely destroyed by large hæmatomas. The cortex is either intact or only affected secondarily. In large hæmatomas the cortex is usually necrotic. The hæmatomas vary in size from that of a walnut to a hen's or goose's egg, or to that of a fist.

They are fixed like a cap on the kidneys, and if the hæmorrhage is extensive, they may surround, compress or flatten out these organs. In extreme cases the suprarenals may be converted into sacs or cysts filled with blood (fig. 81). Hæmatoma of the suprarenal gland is usually a bilateral affection, one side being more affected than the other; if only one side is involved, it is generally the right. The



FIG. 81.—Large hemorrhages of the suprarenal.

hematoma often bursts into the retro-peritoneal tissue, the true pelvis or the abdominal cavity.

Hæmorrhage of the suprarenals is occasionally found in the still-born infant; but it is more usual for death to occur a few hours after birth or on the first or second day. Sometimes death is delayed to the fifth or seventh day; in Morison's case life continued for twenty days. It is not certain whether the disease is absolutely fatal. Theoretically it is quite conceivable that the hæmatoma should gradually vanish after the cessation of the hæmorrhage and that the so-called extra-capsular chromaffine tissue should assume the function of the medullary tissue which has been destroyed. In these cases the post-mortem does not reveal any remains of large hæmorrhages as a secondary condition.

It is very difficult to diagnose hæmatoma of the suprarenal gland during life. There are no signs indicating loss of the chromaffine tissue because this tissue is distributed throughout the body, so that its loss from the suprarenal gland is compensated for. The pigmentation seen in Addison's disease does not occur in the newborn infant, or at any rate, very exceptionally. Rösle observed a patchy pigmentation of the palate in one case, where death occurred on the twelfth day. Magnus thinks that the course of the disease in the newborn is too rapid to permit of the deposit of pigment. He refers to the experiments of Fox which showed that absorption of the suprarenal substance destroys the red blood corpuscles, and he suggests that this accounts for the connection between suprarenal hæmorrhage and icterus neonatorum. But this point naturally does not enter into the question of the diagnosis.

In certain cases the infant is apparently quite well before symptoms of the disease manifest themselves. The disease may begin very acutely, and may be accompanied by fever. The infants refuse nourishment, become drowsy, their breathing is shallow and sometimes there is severe vomiting and convulsions often occur. A rapidly increasing anemia is the most prominent clinical feature, and this renders the probability of an internal hæmorrhage very great. Owing to the large dimensions of some suprarenal hæmatoma, it is quite possible for them to be palpable through the abdominal wall, or their presence may be concluded from signs of pressure on adjacent organs, i.e., the liver and spleen. Death usually occurs in a few days. The end may come in the form of apoplexy, as occurred in a case of Fiedler's, on the fourth day.

Whereas Lissauer ascribes the symptoms of the disease to paralysis of the solar plexus, Materna considers that they are not caused by the loss of vasomotor function, but on the contrary by an excess of adrenin in the blood, or an adrenin toxæmia, resulting from the destruction of the suprarenal medullary substance. It is possible that a congenital hyperplasia of the medullary substance creates a predisposition to it (Rösle). Materna thinks that the cases of suprarenal hæmorrhage which are associated with an adrenin toxæmia can be diagnosed during life (acute onset, rapid

shallow breathing, frequent vomiting, convulsions, tachycardia, high blood pressure, dilatation of pupils), and that these symptoms of sympathetic irritation are probably susceptible to therapeutic influence. In support of this view he quotes a case wherein the blood of the liver had a powerful mydriatic action on a frog's pupil, and the liver cells showed the iron perchloride reaction characteristic of adrenalin. The assumption that the blood was saturated with adrenin was quite justified in this case; but even according to Materna this cannot be demonstrated in all cases.

Cutaneous hæmorrhages, and hæmorrhages in the visible mucous membranes and intestinal tract, may occur in association with suprarenal hæmorrhage.

Suprarenal hæmorrhage occurs with comparative frequency after prolonged labour and in infants who are born asphyxiated. It is quite obvious that asphyxia favours the occurrence of hæmorrhage in the soft tissues. It is also evident that Schultze's method of resuscitation may result in the effusion of blood from lacerated vessels (Hengge). Magnus suggests that the liver may compress the vena cava, causing hyperæmia of the abdominal viscera and effusion of blood into the suprarenal tissue. But these suggestions are not satisfying because, just as in the case of intestinal hæmorrhage, they do not explain the profuse character of the hæmorrhage.

Injury during birth and asphyxia do not completely explain the occurrence of suprarenal hæmorrhage, because this condition may occur after the easiest labour (Dörner, Materna, Morrison, Wellmann, and the author's own observation). Thromboses of the suprarenal veins, which, according to Simmons, is the most frequent cause of suprarenal hæmorrhage in adults, hardly comes into consideration in infants.

There is no evidence to connect these hæmorrhages with any infection. It is, however, remarkable that suprarenal hæmorrhage occurs with comparatively great frequency in the infants of eclamptic mothers (Dienst, Dörner). Magnus has recorded a case of suprarenal hæmatoma in a fully developed stillborn infant, delivered by Cæsarean section, whose mother was an eclamptic. This suggests toxic influence and justifies the inclusion of suprarenal hæmatoma among hæmorrhagic diseases (Ricker).

Hæmorrhages in other Internal Organs.

Hæmorrhage into the cranial cavity and vertebral canal is almost exclusively the result of injury at birth; it is most rare for septic or toxic causes to be responsible for any considerable hæmorrhage in these situations. If any hæmorrhagic disease occurs in an infant already suffering from an intra-cranial hæmatoma, the latter may increase in size owing to secondary hæmorrhage into it. Hæmorrhage into the orbit and within the eyeball, and the frequent ecchymoses of the conjunctiva, are almost exclusively due to trauma and to congestion.

Hæmorrhage in the thoracic viscera is very rare, apart from

the congestive ecchymoses of the pleura and pericardium; but nevertheless considerable hemorrhages have been observed in the pulmonary tissue and the pleural cavity—especially in septic diseases. We have already referred to "pulmonary apoplexy" in connection with sclerema. Shukowski describes a case of hæmothorax in an infant who was probably syphilitic. The infant was suddenly taken ill the second day with signs of internal hæmorrhage and dyspnoea; there was dullness over one half of the thorax. The same author describes a case of hæmorrhagic effusion into the pericardium in a newborn infant due to syphilitic vascular disease.

Severe hæmorrhages do not occur often in the abdominal organs, except in the intestine and suprarenals. The liver is sometimes the seat of small hæmorrhagic effusions, under the capsule and in the parenchyma. Bonnaire and Durante attribute it to toxæmia (e.g., eclampsia) or infection which produces vascular changes and degeneration of the liver cells, resulting in hæmorrhages which are encouraged by the deficient coagulability of the blood. Hæmaturia, as a consequence of renal hæmorrhage, occurs very rarely in the newborn infant. Fabre and Jarricot have recorded two cases of transient hæmaturia, supervening on the third day, accompanied by fever. Syphilitic disease of the vessels may also cause hæmaturia. Hæmoglobinuria and hæmaturia are among the symptoms of the so-called Winckel's disease and of allied conditions, as also of hæmorrhagic sepsis.

Hæmorrhage from the Genital Organs

VAGINAL HÆMORRHAGE.

A discharge of blood and serum, or of blood and mucus, from the vagina of newborn females is occasionally noted in the middle of the first week, usually on the fifth or sixth day, and but rarely earlier. In slight cases, only blood-stained spots are seen on the napkin; in more severe cases blood-coloured plugs of mucus, or shreds or clots are seen on the labia. On separating the latter blood can be seen at the hymen coming from the vagina. The hæmorrhage usually ceases in two or three days; it may last for four to six days, but it ceases absolutely as a rule.

The vaginal hæmorrhage may be a symptom of sepsis (Ritter). Doféris saw four fatal cases among five which came on at the same time. Septic hæmorrhage is rare; it usually comes on in the second week. Vaginal hæmorrhage in the newborn has nothing to do with precocious menstruation. Shukowski thinks that these metrorrhagias depend upon a hyperæmic state of the internal genitals, which is associated with the intestinal hyperæmia consequent upon the irritative condition caused by dyspepsia. Zappert had the opportunity of making a histological examination of the uterus of an infant who had vaginal hæmorrhage. He found that the sub-mucous tissue was permeated by dilated vessels gorged with blood, and that blood corpuscles were exuding in various places. The

epithelium was intact, and there were no signs of inflammation. It is probable that the cause is an increase of a physiological state of irritability, consequent upon the transference of pregnancy toxins (Haltia) from the mother to the foetus. The vaginal hæmorrhage may assume considerable proportions in the course of severe hæmorrhagic disease.

HEMORRHAGE FROM THE MALE GENITALS.

It happens on rare occasions that the urine of male infants contains temporarily some small blood-stained clots. It is difficult to say where the blood comes from. It may come from the vesical mucous membrane, from the prostate, or the urethra. Whipple records a case of profuse hæmorrhage from the prepuce. Severe and even fatal hæmorrhage may follow circumcision (v. p. 460).

Epistaxis.

Hæmorrhage in the naso-pharynx is only recognizable clinically when the source of bleeding is on the surface of the mucous membrane and flows into the lumen. The blood may trickle out of one or both nostrils, or it may flow along the posterior pharyngeal wall into the gullet. If the mouth is carefully inspected the streak of descending blood can be seen. As mentioned previously, these cases may resemble a hæmorrhage from the digestive tract ("false mælena"). Congestion may be a contributory cause of this epistaxis, but there is generally some more serious malady to account for profuse hæmorrhage from the nose. Epistaxis, and especially hæmorrhagic rhinitis, is found most frequently in syphilitic infants, but it is rarely the only symptom. Diphtheritic rhinitis may also be accompanied by hæmorrhage, and there is certainly also a septic form of epistaxis, but in addition to these forms, which are symptomatic, there appears to be an idiopathic hæmorrhage of a profuse character. D'Astros considers that this form of hæmorrhage in the newborn infant is very ominous, but the prognosis must not be considered absolutely fatal.

Hæmorrhage in the Mucous Membrane of the Mouth and the Conjunctivæ.

The palate is often the seat of punctiform and somewhat larger ecchymoses; sometimes the ulcerations on the mucous membrane of the palate, on the median raphe or Bohn's vesicles are surrounded by hæmorrhagic borders. These are congestive hæmorrhages which may become enlarged during the first days of life through the mechanical act of sucking. Should there be any hæmorrhagic disease at the same time, these hæmorrhages may extend superficially and give rise to dark red effusions. Wounds of the mucous membrane of the mouth may bleed so freely that the infant may bleed to death (Greef). Ritter reports cases of septic hæmorrhages from the lips and gums, and Heubner observed a septic case of gummiform hæmorrhage from the tongue.

The ecchymoses which so often occur in the conjunctival membrane of the eye are almost always harmless congestive hæmorrhages. But occasionally superficial parenchymatous hæmorrhage occurs in this situation. O. Müller has observed a case of isolated hæmorrhage from the conjunctiva. The blood kept on flowing from the eyes and formed a cap-like coagulum over the globe. The blood gradually became thinner, but the bleeding could not be arrested. It persisted from the second day to the twenty-fourth, when the infant died. The post-mortem revealed nothing apart from the extreme anemia. Hæmorrhages from the conjunctival sac have also been observed in septic cases, occasionally as a sequel of conjunctivitis.

Umbilical Hæmorrhage.

Umbilical hæmorrhage may come either from the umbilical vessels or from the parenchyma of the umbilical stump. Severe bleeding from the vessels of the umbilical cord is very rare, because under normal conditions the cutting of the cord produces active contraction of the arteries. The powerful longitudinal and transverse muscular layer of these arteries and the deficient development of elastic fibres enable them to contract and to shorten themselves in a centripetal direction (Strawinski and J. Bondi). Possibly the stimulating action of the external air plays some effect in causing contraction of the vessels and the cessation of bleeding from the umbilical cord, but the development of the lesser circulation has a considerable influence, owing to the lowering of blood-pressure which occurs in all the arteries of the aortic system.

The umbilical arteries continue to pulsate for a few moments after birth, not only in the abdominal cavity (Hofmann), but also in the umbilical cord. Although the tying of the cord usually prevents bleeding, the stump requires attention during the first few hours after birth, and if the cord has been cut near its insertion, this attention is all the more urgent. The possibility of bleeding from the umbilical cord is greater if respiration is obstructed and the pulmonary circulation therefore hindered with a consequent rise in arterial blood-pressure owing to congestive phenomena (e.g., in asphyxiated children), or if there are other circulatory defects such as congenital heart disease. Hæmorrhage may also occur even if the cord has been properly tied, if it is very gelatinous, as the evaporation and absorption of fluid may cause the knot to become loose.

Bleeding may occur occasionally from the vessels at the surface of the umbilical wound after the cord has fallen off. In these cases it is obvious that there has been some failure in the normal thrombus formation and obliteration of the umbilical artery in its abdominal portion (Ritter, Granddier, Knöpfelmacher, Althoff). Bondi found that the umbilical vein was remarkably dilated in some infants who died from pulmonary disease at the end of the first week. Whereas the umbilical arteries contract immediately after birth, the veins

collapse without any sign of contraction; if the resistance in the right ventricle increase the collapsed vein may dilate. Possibly this accounts for some cases of hæmorrhage after the umbilical stump has fallen off.

Hæmorrhage from the paræchyma of the umbilical stump is more frequent, especially if the cord has been cut close to its abdominal insertion, or after the cord has fallen off the navel or the granulations in this situation. This hæmorrhage is slight, and ceases after the application of a bandage or after cauterizing.

Hæmorrhages which persist and are difficult to arrest are always very ominous. They indicate that a hæmorrhagic diathesis exists, and the bleeding cannot be stopped, although its source may be quite accessible. They may lead to death after several hours or in a few days (Altkäufer, Lissmann, Granddör, Nohl, Paulsen, Warber). These profuse parenchymatous umbilical hæmorrhages occur in the course of septic disease, in syphilitic infants, or they may occur as a primary condition. The idiopathic hæmorrhages are undoubtedly analogous with melæna and profuse epistaxis.

Whereas severe umbilical hæmorrhage is a very rare event now, it was comparatively common in pre-warrior days. Omphalorrhagia was one of the most frequent symptoms of hæmorrhagic sepsis. In 1875 Knoske wrote as follows in a book on hæmorrhages in early infancy: "The umbilicus almost always participates in the hæmorrhages in infants up to the age of 54 days."

Cutaneous Hæmorrhages.

Hæmorrhages into the cutaneous and subcutaneous cellular tissue, unlike the purpura, which in the hæmorrhagic diathesis of older children is the principal feature of the disease, are secondary in importance in hæmorrhagic disease of infants to internal bleeding. They may often, however, be very extensive. They consist, as a rule, not of dark red flecks as in purpura simplex, but of deep extravasations over an extensive area, of a bluish and glazed appearance. They may occur in various situations—on the sacral region, on the heels, on the occiput, on the chest and abdomen, in the axillary folds, or on the thighs (Holt). Here, where slight lesions are already present, as, for instance, in the neighbourhood of a caput succedaneum, secondary hæmorrhages occur, and extensive bluish extravasations develop in these situations in the course of a few days. In such cases this is commonly followed by deep jaundice and bile in the urine, which is probably due to the large quantity of blood effused (fig. 82). Skin punctures, made for the purpose of a blood examination, often bleed for hours, and a purple blotch forms around this point, exactly as in the hæmorrhagic diseases of older children. Cutaneous hæmorrhages may accompany hæmorrhage in other situations, but they also occur alone. The prognosis in such cases is by no means unfavourable. The signs may disappear in a few days, and complete recovery follow. Warber records a case in which, in addition to severe umbilical hæmorrhage, blood oozed like sweat from the skin folds on the dorsum of both feet.

Hæmorrhage may also occur from the external auditory meatus (Holt).

Hæmorrhages of this kind and hæmorrhagic eruptions may also appear in the course of a septic infection. Septic hæmorrhages and



FIG. 52.—Subcutaneous and subperiosteal hæmorrhage of the skull; hæmorrhages of the eyelids, deep jaundice (infant six days old).

essential skin hæmorrhages can scarcely be mistaken for cutaneous hæmorrhages due to birth-injury and the punctiform congestive ecchymoses so often seen in the face.

The Etiology of the Hæmorrhagic Diseases.

In discussing hæmorrhage from the alimentary tract, it should be remembered that the agent producing the lesions in the walls of the blood-vessels may be mechanical, infective, or toxic. These possibilities also call for consideration in the case of hæmorrhage in other situations. This is to some extent proved by anatomical

and bacteriological investigations, and to some extent only inferred. The classification of individual cases in definite categories according to their clinical features presents many difficulties. Different etiological factors often co-exist; hæmorrhage due to infection, for instance, is probably more liable to occur where a congestive ecchymosis is already present, or where the blood-vessel walls have already been damaged by some toxic agent, and so on.

Apart from the two questions of the nature and cause of the lesion of the vessel wall, the third of the questions already stated requires an answer, namely: Why does the bleeding persist? For this is the essential feature of the diseases under discussion. The hæmorrhages often follow very trifling injuries to the vessels, and apparently occur even in the complete absence of any such injury ("parenchymatous" hæmorrhage). There must be changes in the blood itself or in the vessel walls, which are responsible for the fact that the hæmorrhage continues. It has been observed clinically that the blood in such cases is watery, and appears to coagulate with difficulty, or not at all.

O. Schloss and Commiskey have estimated the coagulation-time in ten cases of hæmorrhagic disease:—

| Coagulation-time during active period of the disease | | | | Coagulation-time after recovery | | Hæmorrhage from skin puncture |
|--|---|---------|---|---------------------------------|---------|-------------------------------|
| | | minutes | | | minutes | |
| 1 | — | 4-50 | — | 4 | — | — |
| 2 | — | 7-30 | — | 3-4 | — | — |
| 3 | — | 9 | — | 8-1 | — | — |
| 4 | — | 5-1 | — | 7 | — | — |
| 5 | — | 6 | — | 3-1 | — | — |
| 6 | — | 3-1 | — | — | — | — |
| 7 | — | 6 | — | — | — | — |
| 8 | — | 10-45 | — | — | — | — |
| 9 | — | 20 | — | 7 | — | — |
| 10 | — | 5 | — | — | — | — |

Abnormalities in coagulation, according to these figures, are decidedly variable. In four cases the coagulation-time was practically normal, in two cases it was slight, in two further cases it was considerably reduced; twice coagulation occurred after some length of time or was completely absent. The secondary hæmorrhage from cutaneous stitches is no constant symptom; it may, in fact, be present, where the coagulation time shows no essential deviation from the normal.

Cases of hæmorrhagic diseases must be divided into two groups. In the first there is delay of coagulation, due to insufficiency or absence of the necessary substances for coagulation of blood. Whipple has been able to prove that there is present in some cases a complete deficiency of one of the most important factors of coagulation, viz., of prothrombin. In a second group of cases the process of coagulation does not appear to be essentially upset. Schloss and Commiskey presume, in such cases, a localized lesion of the vessel wall to be present, which may result in a lack or deficient production of the thrombokinase.

Disturbances of blood coagulation, whether of a general type, or, according to the above theory, with a definite associated vascular area, resemble a disease, in which defective coagulability is the essential feature, viz., hæmophilia. In fact some cases have been described in the literature as hæmophilia neonatorum (Brittin). Larnabee suggests that in the hæmorrhages of newly born children, hæmophilia should only be assumed if there is a family history of "bleeders," or if the children, as soon as they have recovered from the first attack of hæmorrhagic disease, again show signs of hæmophilia. From this point of view he has collected thirty-seven cases from literature, in which the hæmorrhagic disease is probably connected with hæmophilia. Hereditary influences sometimes appear to play a part, particularly in the ætiology of melæna; a few observations have been published of cases of melæna in several children of the same mother and in twins (I. Fischer). In some cases the history has shown that either the father or the mother of the child have shown a disposition to hæmorrhages, which have been very difficult to control (Kosmowski, Salzmunn, v. Wipckel, Waeber). Wittner observed severe hæmorrhages in connection with ritual circumcision; two brothers and eight uncles of the child had died from hæmorrhage connected with this operation.

But it may be considered as proved that in the overwhelming majority of cases that recover, the changes in the coagulability of the blood disappear on the cessation of the symptoms of the disease. The condition is therefore not the first manifestation of true hæmophilia, but of a temporary hæmophilic quality of the blood, which one is accustomed to observe in the hæmorrhagic diathesis of older children. True hæmophilia appears to become manifest only in later life, for the history of such sufferers is mostly negative in respect of hæmorrhages in early infancy. Among 567 subjects of hæmophilia Grandisier only found twelve who had suffered from hæmorrhages during infancy. The fact that newborn girls are as frequently attacked with hæmorrhagic disease as boys contradicts the assumption of a regular connection with true hæmophilia.

Accurate knowledge is lacking as regards the causes of the above-mentioned changes of the blood, and of the nature of the specific toxin which damages the vessel walls. The relatively frequent occurrence of some hæmorrhagic disease (suprarenal hæmorrhages) in children of eclamptic mothers allows one to imagine that possibly poisonous products of metabolism of the maternal organism (so-called pregnancy toxins) are transmitted to the child and thereby cause vascular and blood changes; placental and ovarian substances have generally the quality of producing hyperæmia and hæmorrhage (Halban). Possibly toxic products of decomposition of the infantile organism are also responsible. In chloroform intoxication of pregnant animals Graham was able to produce in the young hæmorrhages, besides cell degeneration and jaundice. He concludes from this, that these pathological changes are to be attributed to general or local deficiency of oxygen, and from this point of view he connects

hemorrhagic diseases of the newborn with a series of diseases of adults (eclampsia, acute yellow atrophy of the liver, phosphorus poisoning, &c.), as well as with those which may occur in the newborn (Buhl's and Winkel's disease, *litterus gravis*). It is of interest, if this assumption is correct, that asphyxia which may act as a mechanical cause of hemorrhage, may also be followed by chemical changes acting in the above sense. One must therefore take care to notice whether a disposition to hemorrhagic diseases exist in those children whose mothers have to be chloroformed for operative measures at confinement.

It must, however, not be expected that one and the same explanation holds good, in detail, for all cases of hemorrhagic diseases of the newborn. Mechanical, infective and toxic factors may provoke such diseases, either alone or combined with one another. The connecting link is to be found in the special disposition of the child during its first days of life, which, in all probability, is based on those changes which the organism undergoes during the transition from the intra-uterine to the extra-uterine life (Baldassari). It may possibly be the manifestation of a toxic effect of those substances which arise during pregnancy in the maternal organism and are transmitted to the child during birth, or such as are formed in the organism of the child itself during the decrease in weight period. Under special conditions such poisons alone suffice to provoke a hemorrhagic disease—in this way, possibly "idiopathic" cases arise—in other cases possibly secondary causes are needed. With such vague conceptions, we are, of course, very much in the realms of hypotheses; nevertheless, this appears to be the right path towards the elucidation of the obscure aetiology of hemorrhagic diseases of the newborn.

Treatment.

In view of the obscure aetiology of hemorrhagic diseases a causal therapy does not lie within our power. There is also no question of prophylaxis. Our chief aim should be to remove the most important dangerous symptom, *viz.*, to arrest the hemorrhage. The arrest of hemorrhage may be local or general. The remedy which has best maintained its place among hemostatic methods is gelatin. According to Moll its effect is chiefly due to an increase of fibrinogen. According to Neu, since the introduction of gelatin therapy the mortality of melena has sunk from 50 per cent. to 13 per cent., according to Noll down to 5½ per cent. The numerous opinions in literature concerning the value of the gelatin treatment are almost unanimously in favour of it (Fulmann, Oswald, Torday, Schubert, Jäger, Meißner, Grüneberg, de Bra, &c.). The gelatin may be administered by enema (200 ccm. of a 10 per cent. solution) or by the mouth (teaspoonful). But the best method is by injection. Merck's preparation is the most universally administered (10 per cent. sterilized gelatin) and is sold in hermetically sealed glass tubes. Most careful asepsis is necessary with gelatin injections. The

gelatin made liquid by warming is injected under the skin of the back, abdomen or thigh, in doses of at least 10 to 20 c.cm. If the hemorrhages continue the injection must be repeated on the same or following day; many a failure may be attributed to too small a dose. Judgment as to the value of a therapeutic measure must be very cautiously given in regard to such a disease that may suddenly and spontaneously cease. A favourable outcome of the disease can therefore only be safely expected by introducing gelatin therapy. But it cannot be denied that many cases seem to behave refractorily towards it.¹

Gelatin therapy has recently been somewhat pushed into the background by another method of treatment, viz., the transfusion of blood or serum of a healthy organism. According to results up to date, the ideal method seems to be the transfusion of blood of the same group. It has been carried out with brilliant success in desperate cases of almost exsanguinated children (Lambert, Sraun, Jacobson, Murphy, Mosenthal, Newell, Lespinasse). A vascular anastomosis is carried out between the radial artery of a healthy adult, e.g., of the father, and a vein (vena femoralis, saphena, jugularis externa) of the child, and the vascular communication is maintained for about twenty-six minutes. The communication of both vessels may be made by means of glass tubes or cannulas which fit into one another (Bernheim, Vincent).

As the transfusion cannot be executed without some surgical skill, simple methods have been suggested. Schloss and Commisker recommend the subcutaneous injection of human blood, taken by venesection 10 to 30 c.cm. at intervals of four to eight hours until the hemorrhage ceases. The serum obtained from the venesected blood can also be injected, 10 c.cm. three to six times daily (Governton). Unger observed good results with smaller doses (2 c.cm.).

Finally animal serum can also be administered. Normal horse serum can be injected or, if this is not at hand, any kind of anti-toxic serum, e.g., diphtheria serum in quantities of 20 to 30 c.cm., possibly two or three times in succession. The result is sometimes highly satisfactory. Green and Swift recommend injections of rabbit serum. Solutions of albumen appear to have the effect of furthering coagulation. Nobécourt and Tixier recommend injections of 5 per cent. solution of Witte's peptone (filtered hot and sterilized). For the first injection 6 to 7 c.cm. and later 3 to 4 c.cm. must be administered.

Too much must not be expected from the internal administration

¹ Up till now there is no account of experiences regarding the recently introduced calcium gelatin (Kalrin, see page 218). The combined effect of calcium and gelatin for hemorrhages should be certainly favourable.

² The administration of umbilical cord serum, recommended by R. Franz, is very useful; it is obtained by centrifugalizing blood collected in test-tubes under sterile conditions and placed in small dark glass bottles, hermetically sealed, and kept for several months in a cool dark place. Amount for injection 20 c.cm. (*Mosch, med. Wochenschr.*, 1912, page 2005.)

of other hæmostatic remedies. These have been used, particularly with gastro-intestinal hæmorrhages. Shukowski suggests the following remedies:

- Liquor ferri perchlor. (two-hourly, 1 drop in a teaspoonful of gruel.—Herschl).
 Ergotin 0.05-0.15 (orally) or subcutaneously.
 Tannin 0.15-30 c.cm. (10-100) (rhassidie, per teaspoon).
 Ext. rhassidie, 0.2-1, ergotin 0.1, ext. stat. 30-50.
 Ext. hydrast. canad., half-hourly, 1 drop.
 Arg. nitras 0.05-100, two-hourly, 1 small teaspoonful.

Calcium is more widely employed (Lægge, Parry)—Cal. chloride, or lactate several times daily 5 to 10 c.cm. of a 1 to 2 per cent. solution;¹ and adrenalin (Holt, Champetier de Ribes and Senleq). The latter may be used in doses of $\frac{1}{2}$ to 1 mg., previously added to an infusion of normal saline.

For hæmatemesis Shukowski recommends washing out the stomach with cold water or normal saline (10³ R.). Absolute rest of the intestine is hardly advisable with hæmorrhages in the digestive tract. Spontaneous recovery has occurred without any interruption of breast-feeding. If every meal provokes hæmatemesis, it is best to apply the child cautiously or to feed it with drawn off milk. If milk at the temperature of the body is vomited, ice-cold mother's milk should be tried in small amounts. Complete starvation treatment might possibly be dangerous, in view of the debilitated condition of some of the children. In any case a sufficient supply of liquid should be given, either by os, by enema or in the form of subcutaneous infusions. The children need rest and warmth. When there is a disposition to subnormal temperatures care should be taken to maintain the normal temperature of the body by means of warm packs or hot bottles.

Local arrest of hæmorrhage only comes into question with hæmorrhages in accessible parts of the body, especially in epistaxis and umbilical hæmorrhage. In the former a solution of adrenalin is instilled or a small wad of cotton-wool, soaked in the latter, or with adrenalin ointment, is placed in the nose. A tampon of tannin gauze or iron perchloride wool can also be applied. The plugging of the throat is not so simple, owing to the smallness of the passage. In umbilical hæmorrhage, if it does not cease with application of the usual styptics, a ligature may be used; though even in spite of this the hæmorrhage does not always cease, or it flows from the puncture channels. The almost harmless genital hæmorrhages need no treatment; if they are manifestations of a general hæmorrhagic disease, local treatment need not be considered.

¹ POSTSCRIPT.—The usual dose given seems to be too small. According to Bigdars ("The treatment of uncontrollable hæmorrhages in infancy," *Berl. klin. Wochenschr.*, 1913, page 142, the body must be positively flooded with calcium salts in order to attain a proper result; the amount administered even to the infant in twenty-four hours should be from 5-6 gm. The easily soluble salts (calc. chloride acetate) are preferable to the lactate and citrate (5 per cent. solution with addition of 1 per cent. gum and possibly 1 per cent. liq. ammonia, zinc, and saccharine).

PART VIII

General Diseases of Obscure
Aetiology(A) GENERAL DISEASES OF OBSCURE ÆTIOLOGY,
COMBINED WITH DEGENERATION, HÆMOR-
RHAGES AND JAUNDICE

In textbooks on paediatrics, among diseases of the newborn, two remarkable conditions are mentioned, which were described several decades ago, viz., Winckel's and Buhl's disease. It is remarkable that these types of disease were only observed very occasionally during the ensuing period, and also the few cases described later under one of these diagnoses deviated considerably from the types of disease described by the first observers. This applies particularly to Buhl's disease. Many cases reported in the literature, under other names, resemble the type of symptoms described by Winckel. They lead to a third group of severe general diseases of the newly born, in which severe jaundice predominates in the clinical picture; they are generally grouped under the term "icterus gravis."

(1) Buhl's Disease.

ACUTE FATTY DEGENERATION IN THE NEWBORN.

This disease, described in 1871 by Buhl and Hecker, shows itself clinically either in the form of post-partum asphyxia which, in spite of the sturdiness of the infant, cannot be relieved and proves fatal, or in the form of a hæmorrhagic disease which develops gradually after the asphyxia has been cured, and with hæmorrhages from the digestive tract, the navel, the nose, extravasations of blood in the skin and mucous membranes, jaundice and oedema, leads to rapid collapse without any remarkable rise in temperature, and usually ends in death before the end of the second week. In typical cases, the initial cyanosis and the disposition of hæmorrhages seem to be absent, and then death sometimes supervenes quite suddenly with rapidly increasing cyanosis. In this indefinite type of disease which can hardly be clinically differentiated from the hæmorrhagic diseases, an anatomical condition has been found

which shows a marked similarity with that occurring in phosphorus poisoning and acute yellow atrophy of the liver; the liver, kidneys and heart show extreme fatty degeneration and are permeated with numerous hemorrhages. In fresh cases the liver is blood red, in later cases it is pale, jaundiced and somewhat more voluminous. Fatty degeneration and hemorrhages are also found in the alveolar epithelium, in the pulmonary tissue and in the bronchi. Occasionally hardly one organ is free from extravasations of blood.

It is very difficult to estimate how far septic or toxic influences are associated with this disease. Range points out a similar disease occurring in newborn mammals, viz., "the foal-halt" of foals, pigs and cattle; it is also accompanied by fatty degeneration and hemorrhages in the internal organs. Roloff attributes these animal diseases to an intra-uterine origin and connects them causally with over-feeding and lack of movement on the part of the mother animals. Range, who has himself described cases of acute fatty degeneration in the newly born, and Knogtelmacher, rank the illness among septic disease. The diagnosis "Buhl's disease" has been seldom made during the fifty years following its first description. Neither the anatomical nor the clinical picture is sufficiently characteristic to render a distinction possible between pronounced septic and haemorrhagic diseases. One solitary pronounced case in recent times has been described by Rüdler as Buhl's disease; it was probably a case of staphylococcal sepsis of umbilical origin, which in the infant of a mother with fever was manifested on the seventh day by hemorrhages, severe jaundice, methaemoglobinemia, degeneration of the organs and had a rapidly fatal termination. The diagnosis "Buhl's disease" might well be replaced by "acute haemorrhagic sepsis".

(2) Winckel's Disease.

CYANOSIS AERIBILIS ICTERICA CUM HÆMOGLOBINURIA.

In the year 1876, during the short period of two months, v. Winckel was able to observe in the Dresden lying-in hospital a peculiar form of disease in twenty-three cases of newborn infants. The children fell ill during the first few days, and relatively most frequently on the fourth day, with pronounced cyanosis, not merely of the lips, cheeks and ears, but also of the trunk and extremities. As a rule jaundice was soon added, which in some cases increased in intensity and in others was merely indicated. The children were lethargic, showed accelerated respiration and increased pulse frequency (though not intense). Only four children survived. The others died, although they were apparently strong, well developed infants. On the average death occurred after thirty-two hours. The shortest duration of the disease was nine hours. Before death, in

¹ POSTSCRIPT.—In a case observed by Luckich with a typical post-mortem condition of Buhl's disease, the bacteriological examination showed *B. coli* bacteriemia. (Festger and Wockensdorff, 1923, page 167.)

many of the cases convulsions set in, and in particular marked twitching of the eyeballs was noticeable. There was no fever; only in one case a temperature of 100° F. was registered. In all cases the most striking symptom was hæmoglobinuria. The sediment of the pale brown coloured urine contained abundant epithelial and granular casts with blood corpuscles, masses of detritus, &c. If the skin was scratched at the most cyanotic places, blackish brown blood, of the consistency of syrup, could be squeezed out. Post-mortems performed by Birch-Hirschfeld showed, besides the cyanosis and jaundiced discoloration of the skin and mucous membranes, a peculiar change in the kidneys. The cortical layer was broader than normal, of a brownish colour and full of minute hæmorrhages, the pyramidal layer was blackish-red with darker stripes (hæmoglobin infarcts). The stomach was considerably dilated and showed numerous hæmorrhages in the mucous membrane. The intestine showed well-marked hæmorrhagic enteritis. In all cases the mesenteric glands were swollen, the spleen enlarged, thickened and tough. In the pleura, pericardium and cardiac muscle there were more or less numerous hæmorrhages. The degenerative changes in the liver and heart were comparatively trifling.

As poisoning by phosphorus, carbonic acid or chlorate of potash was out of the question, the endemic appearance of the disease made the supposition of an infection probable. As, in all cases, the navel was normal, in all probability the poison had penetrated by the digestive tract. The nature of the septic nœxa remained unexplained; the mothers were perfectly healthy; no endemic disease had occurred for months.

The first observers conjectured that it was a disease associated with sepsis. Subsequently an active search was made for a causal organism in similar cases. In a sporadic case Serelitz found streptococci (illness on the eighth, death on the twelfth day), hæmoglobinuria, liquid dark green stools, hæmoglobin infarcts in the kidney, and fatty degeneration of the liver. Finkelschein also found streptococci in a similar case. Within two years Wocynski observed twelve cases occurring in epidemic form with the clinical and anatomical conditions described by v. Winckel. In two cases *B. coli* could be isolated post mortem (Kamen). Sandner observed a case corresponding entirely to v. Winckel's description. Epstein and Ritter also reported similar sporadic cases. In a child of twenty-six days, whose illness corresponded almost entirely with that form observed in the newly born, Francioni found a bacillus similar to a pseudo-diphtheria bacillus. Birch-Hirschfeld had already maintained that the mere presumption of a septic nœxa was not a satisfactory explanation. And even to-day, with the bacteriological findings obtained up to now, the nature of the disease cannot be considered as elucidated. In spite of the relatively small number of cases examined, not less than three kinds of organisms have been found. The question must occur to everyone why such frequent causes of

sepsis as the streptococcus and *B. coli* should so rarely cause Winckel's disease. The question is difficult to answer.

[3] Diseases related to both the above Types

In the literature there is a whole array of accounts of diseases of the newly born, which have shown considerable similarity with the above mentioned types of disease, but have been described under other names. Range mentions an epidemic observed by Bigelow presenting the following course: dark discoloration of the skin as may be observed after the use of silver nitrate, urine containing blood, diptheritic affections of various mucous membranes, and dark green offensive faeces. Apparently the condition is one of haematuria and not haemoglobinuria. In one case the liver presented changes such as are found in acute yellow atrophy.

Parrot observed two cases of a disease, named by him "tubulohæmatis rénale," the symptoms of which correspond almost entirely with Winckel's disease.

Bar and Grandhomme give the name of "maladie bronzée" to a symptom-complex consisting of restlessness, vomiting, convulsions, refusal of nourishment, jaundiced to bronzed colour of the skin, and haematuria.

Nobécourt and Merklin report the case of a child aged ten days that succumbed, after a short period of illness, to severe icterus and cyanosis. At the post-mortem hæmorrhages were found in the liver, meninges and kidneys, the tubuli recti of which were filled with blood coagula. On bacteriological examination of the body all the cultures remained sterile. The authors conclude that the forms of disease associated with icterus and haematuria are not caused by septicæmia alone, but also by intoxications; Knöpfelmacher, commenting on the case, holds firm to the theory of sepsis, in spite of the negative bacteriological findings.

At this point may be mentioned an observation made by the author, according to certain respects Winckel's cases of cyanosis, even if the absence of hæmoglobinuria and the favourable outcome of the disease at once exclude the diagnosis of "Winckel's disease." The affair in question was the repeated occurrence of severe cyanosis among fifteen children in a women's hospital. The cases occurred during the course of a few days, almost simultaneously, in various lying-in rooms and in children of 2-3 days. The symptoms were confined exclusively to an intense cyanotic discoloration of the skin and mucous membranes, which, especially with the jaundiced children, gave the appearance of a tetras. A marked disturbance of the general health was absent, the children mostly drank well, were not feverish, had no dyspnoea and good action of the bowels. On inhalation of oxygen the cyanosis was very little influenced (methæmoglobin cyanosis). After twelve to thirty-six hours the alarming appearance had in every case disappeared. All the children recovered. The type which was noted in most of the cases shared no peculiarities. The epidemic occurrence of the symptoms, which only appeared on this occasion, indicates a common cause. According to the progress of the disease it is impossible that infection was in question. In assuming an intoxication a respiratory toxin must chiefly come under consideration. In contradiction of the influence of the latter, is again the circumstance that the children lay in different rooms, and in the best-arranged apartments of a hospital, satisfying the most modern requirements, and also that only a few children were attacked by the cyanosis. The possibility of a toxin that had entered through the digestive tract seemed more probable. But of what nature this might be, it was impossible to ascertain.

(4) *Icterus gravis*.

Whereas in Buhl's disease degeneration of the organs is the most prominent feature, and in Winckel's disease, and allied forms of disease, hæmoglobinuria, hæmaturia and cyanosis dominate the scene, in a third group icterus, which to a greater or less extent in the above-mentioned diseases is particularly well developed, was present.

Various diseases are connected with Winckel's type, and are differentiated merely by the characteristic symptom, hæmoglobinuria being absent.

Parrot's "*Maladie bronzée hæmaturique*" may be analogous with a type of disease described by Laroque and Charrin under the name "*maladie bronzée hæmatique*," the symptoms of which consist of severe jaundice, green coloration of the stools, and sub-normal temperatures. The authors presume it to be a cryptogenetic infection.

Baumet and Bojadjef have described a case as "*Ictère bronzé hæmatique*" in which vomiting, marked jaundice and twitching of the muscles appeared on the fourth day. The stools and urine were dark, but the latter contained neither hæmoglobin nor one of its derivatives, but indican. After a severe relapse the child recovered.

Lesage and Demelin report on the epidemic occurrence of severe icterus in the newly born within the first week. Jaundice developed after the second day, and increased in intensity to such a degree that the skin showed a bronze colour. Attacks of cyanosis also occurred, the children were mostly fidgety, cried a good deal, and had mild convulsions. The majority of cases were fatal. From the gastro-intestinal symptoms existing at the same time the authorities presumed there was an infection of intestinal origin, in fact *B. coli* was present in the faeces in pure culture, and after death could be shown in the internal organs. Goussard describes a similar case of "*icterus infectieux*" of gastro-intestinal origin; in this case also *B. coli* were isolated after death. The anatomical changes consisted in congestion, hæmorrhages in the liver, occasional hæmorrhages in the cortex of kidney, and a mild intestinal catarrh.

Hüffel isolated a hæmolytic streptococcus from the blood of an infant's heart which had suffered from severe jaundice with bile in the urine and hæmorrhagic diathesis.

Possibly syphilis is associated in many cases with icterus gravis (Rühle, Arkwright).

Apart from the cases of congenital obliteration of the bile ducts, which have sometimes been described as icterus gravis, and apart from the above enumerated diseases, most of which may be of infective origin, there remain cases to be mentioned of severe icterus which have been described as "*habitual icterus gravis*." Plamannstiel, in particular, considers that these cases should be regarded as a specific disease. The most prominent symptom is jaundice, starting very early and increasing in intensity, with

excretion of bile pigment in the urine, without acholia of the stools. The icterus appears either on the first day or immediately after birth (Nahm). Fever is generally absent. Pulse and respiration are not affected. There is a disposition to frequent catarrhal evacuations of the bowels, occasionally mingled with blood, but no marked symptoms of melæna. The taking of food is generally good. The disease is remarkably often associated with meningeal signs of irritation, hyperæsthesia, screaming, tonic spasms of the extremities and back muscles. In the course of the first week it generally leads to collapse and death. It seldom lasts longer, when a hæmorrhagic condition with umbilical hæmorrhages may occur. Relatively often, though not invariably, at the post-mortem examination yellowish discoloration of the nuclei is found, described by Schmidt, Beneke and Esch, consisting of sharply circumscribed yellow coloration of certain nerve nuclei, which is in striking contrast with the usual pale yellow diffuse colour of the rest of the nervous system. The yellow colour affects the lenticular nucleus, Luys' body, the cornu ammonis, the nucleus dentatus, the olive, and, in particular, the sensory nerve nuclei of the medulla oblongata. Esch believes that the bulbar symptoms, existing in many cases (impossibility of swallowing, disturbances of respiration), are connected with these nuclear changes. The anterior and posterior cornu of the spinal cord may show a yellow colour. Furthermore, a considerable enlargement of the spleen is considered as typical, whereas the liver shows no characteristic changes apart from a slight swelling. Fatty degeneration of the glandular organs and the anatomical signs of a septic infection are generally absent. There are often effusions into the serous cavities and the cranial ventricles, catarrh and hæmorrhages of the mucous membranes.

The term "habitual" icterus is used because, in some cases, it is so often repeated among children of the same parents, though healthy children are born in between or only with mild icterus (Pfannenstiel, Beneke, Busfield, Ashby and Wright, Dugot, Lagrèze, Esch, Nahm, May).

As to the ætiology of the disease, most authorities reject the theory of septic factors and a connection with Buhl's and Winkel's disease. Lagrèze and Nahm suggest a congenital intoxication of the fœtus from poisonous products of metabolism of the maternal organism, and May suggests an auto-intoxication from infiltration of the whole organism with constituents of bile, in other words cholemia. Pfannenstiel is of opinion that it is a case of insufficient fitness of the child for independent extra-uterine life, a kind of functional malformation. According to his theory icterus gravis is practically a malignant form of the common icterus neonatorum.

Knöpfelmacher, in a critical review of the cases published up to the year 1910, comes to the conclusion that their course is so similar to those cases proved to be of septic origin, that there is no reason to differentiate them from septic diseases and to give them a new name. The indispensable proof of the absence

of an infection would be lacking for the theory of an individual form of disease. Only in one case of Pfannenstiel's was the bacteriological examination of liver and spleen negative. Even if it is an open question whether infective factors are concerned or not, and whether we are dealing with a symptom complex belonging to the group of sepsis, or depending on auto-intoxication, it is doubtful whether we are justified in assuming a clinical uniformity of the cases. The family histories are based largely only on statements regarding the occurrence of cases of icterus in the family, and cases of death in other children from "icterus." It is a well-known fact that the majority of newborn children are more or less jaundiced during the first few days of life. Very likely the manifold diseases of this period of life may influence the icterus neonatorum occurring at this time in its intensity. It is not uncommon for several children in a family to die during the first few days. Whether it is a common source of infection, a congenital deficient power of resistance against intoxication or infection, or in Pfannenstiel's sense a "deficiency in fitness of the children for independent extra-uterine existence," is very difficult to decide on our present knowledge, even with very thorough clinical, anatomical and bacteriological examination. Any other significance except that of a symptom can hardly be given to icterus.

It cannot be denied that children who die during the first period from some mysterious disease, are sometimes extremely jaundiced. The author observed a premature child weighing 1,800 gm. which after thriving remarkably well at first, after taking the breast for three days, ceased to do so on the fifth day, wasted and died in twenty-four hours. The child had icterus from its second day, which rapidly increased in intensity and gave the skin throughout a dark, oranges-yellow colour. On cardiac puncture made immediately after death, the blood was found to be sterile. The post-mortem proved entirely negative.

Hentner alludes, in his text-book, to the occurrence of mysterious diseases in the newly born, which may possibly be regarded as belonging to the same group as Buhl's disease, the cause of which remains uncertain through absence of post-mortem examination. The case which he adduces as an example was a healthy boy, who stopped taking nourishment on the fifth day, grew rapidly thinner, and with no discernible cause died on the thirteenth day showing extreme quartly, increasing cyanosis and (obviously secondary) diarrhoea.

In such cases the post-mortem does not always offer a satisfactory explanation. On examining the bodies of weakly children that die soon after birth, the pathologist frequently finds degenerative changes in the organs, for which an infection cannot by any means be made responsible. Possibly the so-called "lack of vitality" is often due to changes easily overlooked macroscopically, which make the organs quite incapable of function or they refuse to answer to the demands of extra-uterine life, even in the first days.

If we survey the numerous types of disease given above it will be seen that there are not many features in common, owing to the many transitions between the individual types, which clinically are not distinctly outlined. They are severe and generally fatal diseases, in which sometimes hæmorrhagic symptoms, sometimes icterus and sometimes cyanosis stand prominent. In the majority of cases there is more or less marked degeneration of the internal organs. How this arises, whether primary or secondary signification should be attached to the clinical symptoms, and why one or the other of the above-mentioned symptoms should prevail, remains unexplained. In respect of the ætiology, to a great extent the same considerations apply that were set forth in dealing with the hæmorrhagic diseases. The poison, which is presumably the cause of change in the organs, is certainly of a septic nature in many cases. Apart from the primary septic cases, there may also be those where sepsis is only added to an already existing disease. In other cases it may possibly be less the destructive effect of micro-organisms that reach the general circulation, than the toxins or toxic products of degeneration of the intestinal contents, which arise from bacterial activity. As a third possibility must be considered intoxication or injury transmitted to the fetus through maternal substances.

Much further observation is necessary in the sphere of these mysterious, ætiologically obscure, diseases of the newly born. We need abundant case material, thoroughly observed from a clinical point and careful post-mortem examinations. Only then can one hope for success in scrutinizing and explaining the various types of disease in a satisfactory manner.

(B) TRANSITORY FEVER OF THE NEWBORN.

During the first weeks of life short periods of fever not infrequently occur, for which no cause can be found even on most thorough examination of the child. The fever generally appears on the third or fourth day, sometimes even at the end of the second day; more rarely only on the fifth day. It lasts but a short while, a few hours; sometimes it continues, generally with remissions or intermissions for two or three days, but it hardly ever lasts beyond the fifth day. The maximum temperature is generally between 100° and 102° F.; but with longer periods of fever, temperatures of over 102° F. have often been registered.

The fever can generally be considerably diminished or even stopped by cool packs; though after removal of the cold compresses the temperature frequently rises again. The ultimate decline of fever is somewhat quick and critical (see figs. 20, 21 [pages 108-9], 83-86.)

During the period of fever the condition of individual children varies. Some are remarkably quiet, but scream a great deal at intervals; others give the impression of being lethargic and sleepy. When the fever is of very short duration, the general state of the



Fig. 86.

| | | |
|---------|--|---------------------|
| 1st day | 15 g. | 104 |
| 2nd " | 0 = 0 + 10 + 10 = 20 | 75 g. |
| 3rd " | 25 = 15 + 5 + 60 + 25 + 3 = 108 | 95 g. + 25 com. per |
| 4th " | 5 = 20 + 5 + 10 + 15 + 20 = 70 | 80 g. + 1ea |
| 5th " | 30 = 30 + 35 + 90 + 7 + 60 = about 222 | + 55 com. per |
| 6th " | 20 = 30 + 35 + 75 + 90 = 320 | + 30 " " |
| 7th " | 40 = 60 + 90 + 75 + 10 = 175 | + 60 " " |
| 8th " | 75 = 30 + 50 + 20 + 60 + 45 = 350 | |

From the third day onwards yellow colored stools (1-5-7 times daily). Milk secretion commenced third day. Abundant milk but somewhat poorly suckling breast. Child quiet. Objective findings negative. Jaundice somewhat deep (from the second day onwards). Continent absolute.

child usually presents nothing abnormal. The appetite is generally slight; the children suck badly, even if there is plenty of milk in the breast; the children also take but small quantities of food (tea, nasser, or drawn-off milk) from the bottle. The loss of weight is relatively considerable in the majority of cases.

According to the time of its appearance the fever frequently falls when the lowest level of the weight curve is reached; but the minimum weight is often only reached after the decline of the fever. During the period of fever increase in weight does not occur.

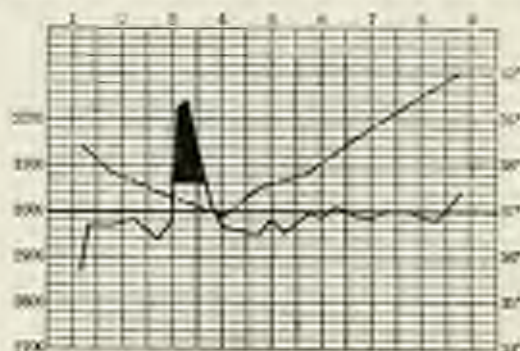


Fig. 87.

Very favorable milk conditions. Milk secretion commenced between second and third day. On the fourth day numerous, slimy, frothy, at first brownish, later yellow green stools (6-7). On the day of fever much increasing; but the child took well. No jaundice. Physical signs negative. Continent absolute.



FIG. 85.

The child took the breast well, but obtained very little in spite of plentiful supply of milk. Refused on the fourth day (— 10 gm.). Extra feeding with drawn-off mother's milk. Stools infrequent (5—6). Obvious underfeeding. Milk suction on the third day. Poorly digesting breast. Rashless during fever, and also later during the night. Extra feeding with tea; No jaundice. Physical signs negative. (1 cold pack). Temperature of mother on the day post part, 100.2° F., otherwise afebrile and normal.

Objective examination of the child shows nothing abnormal; it is a case of "fever without signs." Occasionally the spleen is palpable. The stools present nothing unusual, at least nothing characteristic of the febrile conditions; they not infrequently have the appearance of starvation stools.

The fever does not necessarily attack only debilitated, weakly children under weight, but also strong, well developed children.

What are the causes of these transitory febrile conditions?

It is quite comprehensible that this transitory fever in pre-natal times was attributed to sepsis. At a discussion on "puerperal infection of the newborn" P. Muller stated that Huter, in the



FIG. 86.

The child took the breast at times, 50-60 gm., on the second day. The feeds were first measured on the fourth day (= 32 gm.). On the sixth day no stool, on the third day 3 slimy, brown stools, and yellow stools from the fifth day. Tea was given at night. Very rashless during fever. (1 cold pack). Slight weeping of the base of the navel, otherwise nothing found, jaundice almost absent. Maternal breast full from the fourth day onwards. Confinement afebrile.

year 1852, drew attention to the fact, that just as fever occurs without localization (febricula) in the lying-in woman, similar abortive forms may also be observed in children. E. Quinquand has described these forms more thoroughly. Eriiss also regarded infective processes as the most frequent cause of febrile disturbances, observed by him in the newborn, particularly umbilical infections and their sequelae, and also diseases of the intestine and air passages.

It certainly cannot be denied that Eriiss's theory was right in some of the cases, but it is hardly probable that the transient conditions of fever in the first days of life are really due to general infections with pathogenic germs. It is not comprehensible how a fever, due to umbilical infection, should disappear after a few hours or days without recurring. The same considerations apply to the aetiological significance of ectogenous intestinal infections. An ectogenous infection is chiefly contradicted by the typical appearance of fever on certain days of life. The transitory fever would have to be regarded as an abortive form of cryptogenetic, enterogenous sepsis, acquired during birth. But against such a theory speaks the fact that no kind of coincidence can be established between the fever of the child and any rises in temperature of the lying-in woman.

If, according to the above, one may exclude an infection with foreign, pathogenic germs, as the cause of the transitory fever, there remains yet another possibility existing for the bacterial origin of the fever. The cause might lie in the normal intestinal bacteria, their products of metabolism and resulting toxins. One might imagine that the fight between the myxosium and the milk flora, which occurs at the same time as the rise in temperature takes place, might cause the fever, and that the latter disappears as soon as the milk flora have conquered.

Under entirely physiological conditions the infant is to a certain extent thermolabile. The question must therefore be asked whether the fever is due not only to a hindrance in loss of heat from a high external temperature, or from too warm clothing. The following reasons contradict such an explanation: (1) the fever under exactly similar conditions only attacks particular children; (2) these are by no means so frequently premature or debilitated children, in whose overheating by hot bottles can so easily occur; (3) the rise in temperature always occurs between the second and fifth day; (4) after cold compresses which quickly reduce the temperature, the latter often begins to rise again. The physiological thermolability of the newborn may nevertheless be a factor of some significance in the causation of the transitory fever.

It is remarkable that the fever generally appears between the third and fourth day, in fact on those days which are considered as the most critical time of the newborn period. If one studies the temperature and weight curves side by side, one cannot help thinking that there must be a causal connection between the fever (which

always lies at the angle of the weight curve, generally at the end of the descending limb) and the processes in the organism, which happen during the period of the falling weight.

Holt and Crandall have described the transitory fever in the newborn as *transit fever*. They regard it as essentially the same as that described by Erich Müller as *thirst fever* in diseased infants, a fever that must probably be regarded as a consequence of concentration of fluids. Exsiccation fever would be a more logical description. Heller also is of opinion that the loss of water suffered by the organism during the first day plays an important part. He points out that the higher the rise in temperature, the greater the loss of weight. He found among newborn with—

Loss of weight:—

| Below 100 grs. | .. | % per cent. | feverish children |
|----------------|----|-------------|-------------------|
| 200—300 " | .. | 3.4 | " " |
| 300—400 " | .. | 16.3 | " " |
| 400—500 " | .. | 14.1 | " " |

It is doubtless correct that children with high fever drink less and that the rise in the weight curve and the increase of amount of liquid are frequently coincident with the decline of fever, but the circumstance that fever also occurs in strong children at good yielding breasts, appears to point to the fact that scanty quantities of milk are rather the consequence than the cause of fever. So it appears possible that the concentration of fluids has something to do with the occurrence of fever, but is only a secondary factor. Possibly it hinders the washing out of some kind of specific pyrogenous substances through the kidney, the nature of which is still obscure. It is certainly not out of the question that the products of tissue waste, before their excretion through the urine, especially with deficient diuresis, may have a pyrogenous effect. According to this theory transitory fever must be regarded as a symptom of auto-intoxication. It cannot be a true exsiccation fever because the fever sometimes disappears without the water being retained and without the weight of the body increasing. And if an aetiological significance is attached to exsiccation, then "under-feeding with water," or an external cause, would hardly be present, but much more an exsiccation (from internal causes) independent of the deficient supply of fluid.

The typical occurrence of the fever about the middle of the first week of life permits the deduction that it is causally connected with normal processes occurring in the organism of the newborn at this stage. The various factors, which may be associated with its occurrence, are the following: the replacement of meconium flora by milk flora in the intestine; the irritant effect of bacterial products of decomposition or toxins, and of nutritive ingredients on the intestinal cells, unaccustomed to these irritants, also of the products of absorption on parenteral cells; the presence of products of the breaking down of tissue, such as occurs during the first days of life; the deficiency of water due partly to an inadequate external supply

of fluid and partly to internal causes, and the resulting concentration of tissue fluids and restriction of diuresis; and, finally, the backwardness of the mechanism of heat regulation. The circumstance, that entirely physiological processes are present, though greatly varying individually in their intensity, apparently justifies the theory that the fever, which after all only occurs in a few cases in children, is due to co-operation of several of the above-mentioned factors.

A knowledge of transitory fever is necessary for the medical man in order to prevent him from giving an unfavourable prognosis and using unnecessary therapeutic methods. It is not necessary to state that one should not conclude that every fever occurring during the critical time, is unimportant, and that only a thorough examination permits of the presence of a more serious illness being excluded.

The prognosis of transitory fever is generally favourable throughout; it quickly passes and leaves no sequelæ. The treatment is purely symptomatic. Cool compresses, which tend to reduce the temperature to the desired level, are much to be recommended. As regards feeding, exactly the same rules apply as in non-feverish children. It goes without saying, that under conditions of exiccation, the proper amount of fluid must be administered.

PART IX

Infectious and Septic Diseases

INTRODUCTION

WHEREAS the newborn child shows as much susceptibility to many infections as an older child, its resistive power towards a large number of morbid agents, particularly towards septic micro-organisms, is considerably less than in later life. But to many infectious diseases the young infant seems to be immune or is extremely rarely attacked by them. The relatively high susceptibility of the newborn child for septic diseases can easily be understood, since its delicate organism exhibits a certain insufficiency even as regards the physiological stimuli of extra-uterine life. It can be understood that its means of defence against infective agents also are still incomplete; the absence of regional enlargement of the lymph glands in local infections is a striking clinical expression of this. Possibly a deficiency of "bacteriolysins" plays some part in the development of the septic processes; Gundobin refers to the researches of Klinoff, which showed that when pure cultures of staphylococci were treated with umbilical cord serum—in contrast with other bacteria that were killed—only a short cessation of growth was noticeable.

It is much harder to understand the peculiar resistance of the young infant, e.g., such as is apparent towards scarlet fever and measles. The organism during the first period of life must be in command of protective measures that are gradually removed in the course of growth. A natural resistance of the newborn might be due to the presence of anti-bodies which exist in the organism of the foetus naturally. These anti-substances may have no peculiar antigen character, a resistance which might be caused, not through specific immune substances, but is founded on the general chemical composition of the fluids of the body. The researches in respect of the biological relation of exchange between mother and child, which have proved that foetal and maternal substances affect each other as antigens, have taught us that to a certain extent mother and child are biologically antagonistic to each other "like two different individuals" (Halban, Pfäundler). According to this train of thought it would be conceivable that the resistance towards one and the same disease would sometimes vary with the child and might be greater than with the mother. The foetal placenta represents, according to Pfäundler, the boundary organ, which neutralizes the

toxicity of the nutrient material coming from the mother. A passage of material which is foreign to the fetus only occurs with a lesion and relative insufficiency of the placenta. Only thus can one understand that in a severe disease in the mother the child entirely escapes. This does not apply only to many infectious diseases but also to malignant new growths. Joseph points out that in such cases the fetus generally escapes the maternal disease, even if the body of the pregnant woman is positively riddled with sarcomatous or carcinomatous metastases. (He only mentions one, a solitary case in medical literature, of a carcinomatous metastasis of the knee, described by Friedrich, in the fetus of a mother suffering from cancer of the breast.)

Apart from a natural resistance, which must be already present in the embryonic rudiment, but is otherwise independent of the parents, the absent or only trifling susceptibility of the newborn child to many diseases, may also be derived from an artificial immunization, proceeding from the mother. According to Wegelius the following causes may produce this immunity:

(1) It may be caused by hereditary transmission of those factors, which produce immunity in the parents, to the germ plasma, so that these factors are also maintained in the progeny of the future.

(2) By the effect of agents, which cause the reaction of immunity in the mother, on the germ plasma or on the fetal organism—active intra-uterine immunization.

(3) By transmission to the fetus of anti-bodies formed in the body of the mother—passive intra-uterine immunization—and

(4) By transmission of anti-bodies to the progeny whilst suckling.

Actual inheritance of immunity or transmission through the germ plasma apparently does not occur (Ehrlich, Ehrlich and Hübener, Vaillard, Wernicke).

Active immunization of the fetus *in utero* is only possible if the respective antigens reach the child, through an inefficient placenta, and the latter shares the disease, or if such penetrate the fetal placenta and cause formation of anti-bodies (Pfaundler). If such a process of immunization occurs at all it is, at all events, very rare. But its theoretical possibility must be admitted, as intra-uterine transmission of diseases undoubtedly occurs; it is very doubtful whether active immunization plays an essential part in the immunity of the newly born. Wegelius found also in active immunization of the mother a marked passive immunity in the child. But this is not to be attributed to the fetal tissues not being capable of reacting on the supply of antigen by the formation of anti-bodies—this ability has been proved by the researches of Kreidl and Mandl, as well as Wegelius—but probably to the fact that the intact placenta is not permeable by dissolved and formed antigens. Ascoli was able to prove, that after introducing heterologous albuminous bodies into the maternal organism, they can only be found in the fetal serum if large amounts are injected, and that the reactions in the maternal serum are always stronger than in the fetal.

A transmission of anti-bodies through the placenta or a passive immunization of the foetus seems to be much more likely. But experimental results in this sphere are certainly not uniform. According to Behring the normal placenta, as a dialyser, is very difficult for high molecular bodies to traverse; and it does not permit the passage through it of maternal anti-bodies. This theory is supported by the negative results of experiments by Römer, Reindlinger, Kasel and Mann, Dzierzowski, &c. They dealt with the anti-bodies of diphtheria, tetanus and typhoid which were not transmitted to the foetus either by active or passive immunization of the mother. But on the contrary, there are results of experiments which proved the actual occurrence of a placental transmission of anti-bodies (Jurewitsch, Stäubli, Capaldi, Rostotski and Funck, Dieudonné, Schumacher, Polano). An intra-uterine transmission has been proved, not only of anti-toxins and agglutinins, but also of haemolysins and precipitins (Merkel). Even if the occurrence of placental transmission of anti-bodies must be unreservedly admitted, the question whether we have to deal with a physiological function of the placenta must not immediately be answered in the affirmative. Plauder believes that the positive results of experiments are to be traced to special conditions created by experiment or spontaneous disease, which are responsible for the transmission; he does not regard the transmission of anti-bodies from the maternal to the infantile blood as a physiological function of the placenta. But Wegelius, who experimented with tetanus toxin, vibriolysin and *B. coli*, came to the conclusion that when anti-bodies were present in the serum of a pregnant animal, they were not only always to be found again in the young, but showed a higher anti-body *titre* of serum than the mother animal; he concluded from this that the transmission of anti-bodies from the mother to the child was not to be regarded as a simple process of filtration, but that an elective power must be attributed to the placenta in respect of the substances which are to be found in the maternal serum. These conditions do not quite coincide with the results of analogous experiments of other investigators. Thus, Schenk found that the proportion of agglutinable substance and serum alexin in the maternal blood is always greater than in the corresponding infantile blood. The antitryptic power of the maternal serum is also greater as a rule than that of the child's serum (Mohr). It may safely be assumed that the various protective materials and anti-bodies are not transmitted in the same proportion. The anaphylactic reactive bodies appear to be particularly easily transmitted to the newborn by the placenta (Rosenau and Anderson, Schenk), but it is not yet known how far this important condition, proved in experiments on animals, is of significance for human pathology.

Our information in respect of the transmission of anti-bodies through the milk are founded on Ehrlich's famous experiment with suckling animals. He made female mice conceive about the same time. Half of these he treated with abrin or ricin to

immunize them, the other half he left without treatment. After the birth of the young he exchanged them, in that he let the young of the normal mothers suck from the immunized mothers, and the young of the immunized mothers suck from the normal ones. In the latter he found, after a little while, a somewhat rapidly diminishing degree of immunity. In the young of the normal mothers, and therefore born free from antitoxin, but reared by the immunized mother, he was able to detect immunity developing during suckling. By many further researches it was demonstrated that tetanus antitoxin and diphtheria antitoxin can be transmitted by the milk (Ehrlich and Brieger, Ehrlich and Wassermann, Brieger and Cohn, Römer, Salomonson and Madsen, Veillard, Sobotta, Schmid and Pfanz, &c.). In respect of the transmission of other antibodies in the milk, positive results in experiments have been obtained for agglutinins (Schumacher, Staab), hemolysins (Bertazzoli), opsonins (Eissler and Sobotta), bactericidins (Schenk). Römer points out that a transmission of bactericidal capacities to the infant may take place through the maternal milk, even though bactericidal effects cannot be shown in the milk. It suffices if one of two bactericidal factors is present in the milk and afterwards the complementary component is found in the infant's blood. Kleinschmidt was able to prove the presence of a bactericidal antiseptor in the mother's milk.

CHAPTER I.

ACUTE INFECTIOUS DISEASES.

Variola (Vaccination).

The susceptibility of the newborn child to small-pox appears to be somewhat great. According to Bollinger, in small-pox epidemics the mortality in children is greatest during the first year of life, and among these at its highest in the first month of life. Curschmann mentions that among children born in small-pox areas he did not see one escape the disease, but that some children only showed very slight development of the exanthem. Willigen also observed five die of small-pox among eight premature children whose mothers were suffering from small-pox at the time of their confinement.

Little is known of the clinical course of the disease in the newborn child. Since the general introduction of cow-pox vaccination variola has become very rare, and an epidemic appearance of small-pox is hardly ever now observed in civilized countries. The malignancy of the disease, however, does not appear to be less in the infant than in later life.

The variola of the pregnant mother often leads to abortion and premature birth. Several observations have been published of intra-uterine transmission of small-pox, but the occurrence is

generally declared to be very rare. The child may be born with scars of small-pox, and thus, with signs of an extinct disease, or in the eruptive stage.¹ The pustules are supposed sometimes to have a different appearance in the newborn, as desiccation is prevented by their being continually soaked in the liquor amnii. In a review of the literature, Kehler differentiates the following four possibilities in respect of placental transmission of variola:—

"(1) The fetus is only born some weeks after the subsidence of maternal variola with fresh pocks.

"(2) Pregnant women, in whom small-pox infection is out of the question, or who are immune, through vaccination or re-vaccination, or who have had mild attacks without any rash, may bring into the world children with a typical variola eruption or scars of small-pox.

"(3) Of twins coming from different ova, one may be born with small-pox pustules and the other may be perfectly healthy.

"(4) The child, whose mother has suffered from small-pox during pregnancy, may be born without a trace of variola, and prove immune to vaccination."

The course of vaccination in the newborn and young infant is different in a certain respect from that in the older child, whether the inoculation be undertaken with humanized or animal lymph. The most striking difference consists in the constant absence of inoculation fever (Behm, Gast, Wolff). Also the local progress is different in so far as severe signs of reaction are generally absent in the neighbourhood of the vaccination pustules. Glandular swellings, otherwise a frequent occurrence after vaccination, do not as a rule appear in the newborn (Klotz). The recommendation, by many authorities, to vaccinate the children as early as possible, even during the first few days, is fully justified by its mild course.

The relatively frequent occurrence of unsuccessful vaccination in the newborn, described by many writers, is most remarkable (Ablass). How far, also in the variation of the course (retarded progress, accelerated reaction [see v. Pirquet]), not only the small-pox of the mother, but also vaccination of the father before or during pregnancy, is of influence cannot be determined at present, owing to the lack of a sufficiently large number of cases. The researches on the possibility of an "intra-uterine inoculation" are chiefly supported by the difficulty of "taking" of vaccination in children of inoculated mothers. Thus, Burckhardt observed, after subcutaneous inoculation of two mothers, that in both children the vaccination remained unsuccessful, whereas two control children showed splendid inoculation pustules. Palm observed in children of mothers who had been successfully vaccinated during the last four months of pregnancy, that the vaccination was always followed by

¹ Forrester, — Artz and Kertl, and also Forrester have recently reviewed cases of congenital small-pox eruption and congenital small-pox scars. (*Wiener klin. Wochenschrift*, 1913, pp. 787 and 795.)

formation of pustules, but that the latter generally showed a retarded progress and did not reach the same extent as those in children of non-vaccinated mothers, and that in one case the vaccination had to be repeated four times before the formation of pustules was produced.

It has not hitherto been stated in the description of the course of vaccination whether the children of vaccinated and non-vaccinated mothers showed any essential differences. But it is worthy of notice that hitherto, in all vaccinated newborn, the progress of inoculation was different from that in an older child, so that apart from an allergy of the child, transmitted from the mother (vaccinated or immunized through small-pox) the possibility must also be recognized, that the newborn organism reacts differently to vaccination from that of an older individual.

Varicella

The long incubation period of variola (two to three weeks) points to the fact that newborn children, even if they may be susceptible to the contagion, do not fall ill during the first week of life, and intra-uterine development of varicella only very rarely occurs.¹ That a certain degree of susceptibility is present during the first few months of life is proved by Sperr's statistics concerning infectious diseases observed during the years 1905 to 1912 in the out-patient department of St. Anna's hospital in Vienna; among 185 cases of chicken-pox four were in children in the first month of life.²

Morbili

That there can be intra-uterine transmission of measles by the blood cannot be doubted after the relatively numerous observations published (Thomas, Gautier, Ballantyne, Fiori, Lomer, Kien, Laur, Moser). If a pregnant woman develops measles, very often labour pains set in, resulting in an abortion or premature confinement. In the majority of cases the birth followed immediately the appearance of the eruption, or a day or two later, more rarely at the end of the maternal illness. The children generally showed the same state of disease as the mother, and were born therefore with the eruption fully developed. The focus in *utero* is therefore, in such cases, infected simultaneously with the mother. Occasionally the illness of the child may follow that of the mother, so that the latter is already in the stage of desquamation, while the child shows a recent eruption. In a case observed by Kohls the exanthem appeared in the child five days after confinement—being the eighth day of the mother's illness; the intra-uterine infection followed in this case the commencement of the maternal illness; the incubation period of thirteen days to the appearance of the exanthem could be exactly proved.

¹ Postscript.—Priddy describes a fully-developed varicella exanthem in the first few hours of life. (*Brit. Med. Journ.*, May 17, 1913.)

² Published by kind permission of the medical superintendent, Dr. Sperr.

In Laur's case the mother developed measles the day after confinement, and the child on its eighth day. Whether this was a case of intra-uterine or air-borne infection is difficult to decide. The author presumes the former. Moser described a similar case: confinement before the disappearance of the mother's eruption, the child fell ill after fourteen days.

Measles in a pregnant woman need not always be transmitted to the fetus, even if birth occurs during the illness of the mother. The children may be born without measles and escape the disease altogether, even if they are suckled by the mother (Gautier, Salus).

It is relatively rare for a pregnant woman to develop measles, as most women have had measles in childhood and are consequently immune.

The newborn child and the young infant prove themselves, as a rule, insusceptible to measles until about the second month. Children in the first month rarely contract the disease, even if they are exposed to infection, such as may often occur with simultaneous illness of the brothers and sisters. Can the newborn child be insusceptible to the common air-borne infection when the fetus can be infected through the blood? Pirquet answers this question in the negative and refers to the numerous cases of measles in quite young infants (forty-seven children in the first month of life) during the epidemic in Ireland, which occurred in 1862 after an interval of four years and attacked a large number of the population that had not yet had measles. It appears, therefore, that only in regions with regular epidemics of measles the newborn remain insusceptible. A child, whose mother has never had measles, is possibly susceptible to the disease from the moment of its birth, just as it may be infected before birth through the placenta. The insusceptibility of young infants is probably due to a passive immunity transmitted by the mother, which is retained for several months, but it is by no means out of the question that there exists a certain degree of natural immunity of the newborn to measles infection.

We have no detailed information with regard to the course of the disease acquired *in utero*. As those infected are generally premature children the prognosis is more serious than in healthy infants, in whom measles frequently has a very mild, abortive nature.

One should be very cautious in diagnosing measles in the young infant. It can only be made with certainty, if, besides the rash, thoroughly characteristic symptoms are present (Koplik's spots, leucopenia); in infants there are so many "morbillous" exanthemata that the opportunity is frequently given for a false diagnosis. It is not unlikely that many cases have been frequently wrongly diagnosed as measles in the newborn.

Since in our country most mothers have had measles in childhood, for this reason in practice we may take the insusceptibility of the newborn child for granted and draw our conclusions accord-

ingly. If the brothers and sisters develop measles or are in the stage of incubation, strict isolation of the newborn child may be omitted; the probability that it will escape the disease is about 100 per cent. But should the exceptional case happen that the mother develops measles after confinement, the question arises whether she should suckle or not. Separation of the child from the mother, even with susceptibility of the infant, is not of much use, as the latter would already have been infected, when the diagnosis is made in the mother. Measles, therefore, is no contra-indication whatever to suckling.

Rubella.

Sporadic German measles, even in older children, can seldom be diagnosed with absolute certainty. All the more caution must therefore be given in the diagnosis of such cases in the first weeks of life. Schick quotes accounts of German measles in a newborn child (Scholl) and a fifteen day old child (Shukowski) but emphasizes the fact that it is extremely difficult to diagnose with any certainty German measles in so young a case. During the rubella epidemics in recent years, not a single infant, among the huge out-patient maternity section of the Vienna Children's Hospital, could be observed with unmistakable German measles. Apparently the susceptibility of the newborn child to rubella is either absent or very slight.

Scarlatina.

According to the literature intra-uterine transmission of scarlatina from the mother to the child is possible (Ballantyne-Milligan, v. Winckel). The diagnosis in this case is still more difficult than with measles. It is well known that in pregnant and lying-in women, during the course of septic diseases—especially of streptococic infections possibly closely allied to scarlet fever—scarlatiniform exanthemata occur; the assumption that it is true scarlet fever is largely a question of the opinion of the observer at the time.

It, in addition to signs of general infection, the child shows a rash resembling scarlet fever, still more caution is needed with the diagnosis than in the case of the mother. Every paediatrist knows that in the infant, besides morbilliform exanthemata benign scarlatiniform rashes are not at all uncommon. It is a fact that the morbidity and mortality from scarlet fever are remarkably slight during the first months of life. The youngest child observed by Escherich and Schick to contract scarlet fever was four months old. The frequent reports of scarlet fever in the newborn are mostly due, in the opinion of the above authorities, to diagnostic errors. Without doubt, typical scarlet fever in children of the first week is most exceptional. Pospischil observed a case of scarlet fever in a child of seven days.

The low susceptibility of young infants to scarlatina is illustrated by Salge's case: a nurse with scarlet fever suckled her child aged

one month during the disease, without the latter becoming ill or its thriving prejudiced in any way. It may safely be asserted that scarlet fever in the mother presents no contra-indication to suckling as far as the child is concerned.

Diphtheria.

True diphtheritic diseases in the newborn have often been described. In the old Vienna foundling hospital Schlickeher observed in the course of a long period fifteen cases of diphtheria among children aged six to twenty days, which in all probability were causally connected with each other and could be traced to the same source of infection. Yet the disease attacked, almost exclusively, weakly, sick children. The healthy newborn child appears to possess a certain resistance against infection from diphtheria, so that the latter may be considered as an uncommon disease in the first weeks of life. This may be due to a specific immunity inherited from the mother, which finds its expression in the almost constant presence of protective bodies against diphtheria in the organism of the newborn. v. Groer, K. Kassowitz and Schick obtained, in their examination of newborn, by means of Schick's intra-cutaneous reaction, almost entirely negative reactions to diphtheria toxin (about 84 per cent.). Fischl had already proved earlier that the blood from the umbilical cord shows antitoxic and bactericidal properties to diphtheria toxin and to the diphtheria bacillus.

Up to date the youngest observed cases of diphtheria established clinically and bacteriologically were those of children aged six to eight days (Röthler, Wolkstein, Auden). The diphtheria may be situated on the tonsils, and occur in an ordinary way in well-formed whitish membranes.

In a child of ten days Sattler was able to find typical diphtheria of the tonsils; the infecting bacilli were derived in this case from a crack in the maternal nipple, which probably received the infection from the throat of an elder brother recovering from diphtheria.

Primary diphtheria of the nose seems to be relatively the most frequent, but there are probably other non-diphtheritic croupous rhinitides (Forrest, diplococci); it is certainly not justifiable, with purulent rhinitis, even if blood is mingled with the secretion, to regard the disease as diphtheria, merely on the basis of microscopical and cultural presence of bacilli—the occurrence of non-virulent diphtheroid bacilli, and of pseudo-diphtheria bacilli in the nasal secretion, is well known. The disease may be caused by epithelial injuries to the nasal mucous membrane (Christeana and Brückner); care must be taken again to avoid confusion with so-called "pseudo-diphtheria," an affection of the soft palate, sometimes accompanied by formation of membrane. The diphtheritic process may attack the pharynx, the œsophagus, even the intestinal mucous membrane (Röthler). Involvement of the larynx (croup) appears to be extremely uncommon in the newborn. A case of true diphtheria of the conjunctiva (together with diphtheria of the nose, throat and

palate) was observed by Forest. True diphtheritic diseases have been observed in the navel (see page 431) and in the penis after ritual circumcision.

All authorities are agreed that the prognosis of diphtheria in the newborn is very serious. If there is reason to suspect diphtheria, immediate treatment with diphtheria antitoxin is indicated. Not less than 150 units should be injected.

If a mother who is suckling her infant contracts diphtheria, the greatest caution must be used, in the child's interest, and the possibility of its infection must be taken into consideration. Perhaps the above-mentioned Schick's reaction is indicated in this respect, in order to confirm or exclude the danger to the child. There is no cause to take the child from the breast, unless the diphtheria in the mother is so severe that in her own interest the suckling should be discontinued. A child susceptible to diphtheria may be protected from infection by a prophylactic serum injection. It goes without saying that the child, in the intervals between the feeds, should be removed from its mother.

Epidemic Parotitis

Non-purulent contagious inflammations of the salivary glands occur very rarely in the newborn. In the literature a few cases of mumps have been recorded, e.g., by White (illness on the sixth and seventh day during an epidemic of mumps), Gantier (submaxillary swelling in a newly born, twelve days after the mother had developed parotitis), Demme (parotitis in the third week of life), Homanns (unilateral, painful parotid swelling, lasting some days, in the child of a woman who contracted mumps a few days before her confinement). As a rule, mumps belongs to those infectious diseases for which the newborn child seems to be very little susceptible. In Sperr's statistics there are only two cases of mumps in infants; one of these was in the first month of life.

Pertussis.

Whooping-cough is also very uncommon during the newborn period, but there is the question whether low susceptibility is responsible for this, or, more probably, the circumstance that only very seldom is there an opportunity of infection. According to G. Seicker the few cases of whooping-cough during the first few days are the following: Bouchet records the case of a newly born that was infected on its second day, began to cough on the fourth day and on the eighth day fully developed pertussis. In Watson's case the child manifested whooping-cough on its first day, its mother during the last weeks of pregnancy having had with her a child suffering from whooping-cough; Killist and Barilez observed a child, whose mother had suffered from the disease during the last month of pregnancy, develop violent attacks even on the first day.

After the expiration of the first two weeks, during which whoop-

ing-enough is only very exceptional, one sees children more or less frequently suffering from pertussis. Therefore brothers and sisters with whooping-cough should be isolated as much as possible from young infants, perhaps especially so, for the younger a child is the more are lung complications to be feared, as they tend to terminate fatally. If the rare case does occur, that a mother who is suckling is attacked by whooping-cough, the greatest precautions should be taken. Whether it is true pertussis, or only attacks of coughing resembling pertussis, the mother should be warned to be as careful as possible not to cough on the child when suckling. The wearing of a face mask is to be highly recommended in such cases.

Influenza.

The last mentioned precautionary measures, which appear to be indicated with pertussis in the newborn child, apply also to influenza and diseases of the air passages of the nature of influenza (grippe) to which the young infant is highly susceptible (see page 201).

During epidemics of influenza in lying-in institutions, newborn children have been observed, not only with catarrhal diseases of the air passages, but also with disturbances in the digestive tract (enteritis), and delayed healing of the umbilical cord (Möller, Chamberlen).

Influenza in pregnant women sometimes leads to hemorrhages into the foetal membranes and to a premature end of pregnancy. In the organism of the child it appears to cause serious changes. Kockel and B. Fischer report cases of congenital cardiac defects, following foetal endocarditis, which in one case was traced to acute bronchitis in the mother two months ante partum, and in another case to influenza which the mother had had six weeks before her confinement.

Typhoid Fever and Allied Diseases.

Typhoid fever in the infant generally runs a very mild course and frequently assumes the form of intestinal catarrh. It is not known whether true, acquired typhoid diseases occur during the first weeks of life.

Typhoid in the pregnant woman may lead to abortion and premature birth or to infection of the foetus. Bacilli have been repeatedly found in the foetus. Apart from slight swelling of the spleen, there are no specific typhoid changes (Morse, Ernst, Dürck, Frenkel and Kiderlen, &c.). Hemorrhages or other pathological changes of the placenta appear to facilitate the passage of typhoid bacilli into the foetal circulation. In a case of Ernst's the pregnant woman had had an injury a week before getting typhoid; in the second week of the disease she bore a child, that after four days of artificial feeding fell sick with severe jaundice and an exanthem, and died after a few hours; the infant's blood

contained enormous numbers of typhoid bacilli. Maternal typhoid may, however, have no ill effect on the fetus. Kehrer and Chamberlaine report cases in which children were born healthy and at the normal time. Possibly in such cases, in which the children escape infection, protective materials pass into the fetal organism. That typhoid agglutinins can be transmitted to the fetus by the placenta has often been confirmed (Jehle, &c.); at least in the second half of pregnancy this appears to be the case. If the typhoid occurs during the first period of pregnancy, the fetal serum shows no specific agglutinating capacity.

Intra-uterine infections of the fetus occur with other bacteria of the col-typhoid group. The intestinal contents of the child of a woman suffering from dysentery, and who died two hours post partum, showed at the autopsy swelling and redness of the intestinal mucous membrane, from which Marckwald cultivated Kruse's bacillus. Nagwerk and Flinzer have described an intra-uterine infection with a paratyphoid bacillus, which led to symptoms of melena (see p. 450).

Tetanus.

Whereas in the majority of the infectious diseases mentioned above, the susceptibility of the newborn child is small, whether owing to a natural or passive immunity transmitted by the mother, it may be accepted that tetanus infection easily finds an entrance into the organism of the newly born. As expressed by the commonly used term "*tetanus neonatorum*," it appears that particularly good opportunities for the development of tetanus are afforded during the newborn period. This may be explained less by the special disposition of this period of life, than by the circumstance, that tetanus bacilli find a suitable focus for development in the physiological wound of the navel and in the lifeless remains of the umbilical cord.

The tetanus bacillus is not only an inhabitant of garden soil, but is also found in the house dust, between the boards, in the furniture, &c., and with dirty hands or instruments may easily be transmitted to the umbilical cord or wound. From thence it disperses its toxins along the paths of the motor nerve trunks, settles in the motor cells of the spinal cord and medulla oblongata and causes enormous increase of excitability of these centres.

The tetanus bacillus has sometimes been found to be in the secretion of the umbilical wound (Baginsky, Kitasato, Peiper and Beumer, &c.) but this is not so in all cases, so that the bacteriological finding cannot decide the diagnosis. Escherich recommends, for the confirmation of the presence of the bacilli, the use of cultures and the scratching of the umbilical wound with a sharp spoon, and the inoculation of mice with the tissue obtained.

If tetanus neonatorum is nowadays a rarity, this is chiefly due to the careful treatment of the navel, the importance of which is well known even to none too "aseptic" midwives. Where this

is not the case, the morbidity and mortality from tetanus in the newly born is enormous. According to Miron's statistics (for the year 1904) of 23,398 children who died in Roumania within the first month, 16,257, or nearly half, died from tetanus. According to Paulin (1906) in Denmark fifty to sixty children die yearly from tetanus. Tropical medical men report that in Cayenne 16 to 25 per cent. of the newborn succumb to tetanus (Flesch); also among the black population in South America tetanus neonatorum is supposed to be very widespread (Anders and Morgan).

The incubation period of tetanus varies. It may be one and a half to two days, but also longer, sometimes weeks. The onset of the disease generally occurs at the end of the first or in the second



Fig. 87.—Tetanus.

week, more seldom in the third; but cases of tetanus have been observed which occurred still earlier, even on the day of birth (Heubner).

When fully developed the symptoms of the disease are very characteristic and generally allow of a diagnosis at the first glance. Sometimes the symptoms of spasm are preceded by a stage of restlessness, lasting several hours, sometimes with loud screaming. The first striking symptom is trismus. At first the child will seize the nipple, but presses it firmly between the gums; very soon it cannot be induced to suck. Every effort to administer food, either from the breast, the bottle, or by means of a spoon, is accompanied by a fresh exacerbation of the jaw spasm. It is finally no longer possible to open the jaws, as the latter are so pressed together, owing to the rigidly contracted masseters, which to the external

touch are like tight strings or ridges. Besides the masticatory muscular apparatus, the facial muscles are also attacked with spasms; the face assumes a peculiar and characteristic expression; the eyes are closed, the forehead wrinkled, the corners of the mouth are distorted, as it were, into a smile (*risus sardoniacus*), the mouth sometimes protrudes like a snout. The spasm soon attacks the rest of the body. The upper arms are pressed tightly to the trunk, the hands are tightly clenched, the legs are half flexed at the hip and knees-joints, the muscles rigid. The abdominal walls, muscles of the back and neck are stiff and rigid. The entire musculature is in a state of extreme *sarri rigidity* (fig. 87). When the respiratory musculature also participates in the spasms, the diaphragm contracts in jerks, and cyanosis occurs. The tetanus is not continuous, but occurs in attacks, as though a strong electric current was passed, from time to time, through the body. Every effort to administer food, in fact any manipulation of the child, even touching the feet or any kind of thermal stimulation, produce afresh the violent spasms. At the height of the attack, the child may be lifted by the feet or head, just like a wooden doll.

The intensity of tetanus is not always the same. There are doubtless mild forms in which the spasms are confined to the masseters, the facial and cervical musculature; in these strong external stimuli are needed to cause general spasms, or they may be absent altogether. The disease may pursue its course with very high temperatures (104° to 108°), but also without any fever. Frequently fever exists with deep intermissions, so that daily fluctuations in temperature of 2° F. may be registered.

Tetanus may lead to death within a few hours or one or two days. As a rule it lasts several days. Its course may be protracted, especially in those cases that recover.

Statements regarding the prognosis of tetanus neonatorum vary considerably. Whereas, e.g., Shukowski estimates the mortality as 98 per cent., according to Frome it is only 41.66 per cent., and Fleisch also does not regard the prognosis to be so bad as is generally considered. For determining the prognosis he gives the following points:—

- (1) Length of incubation period (generally difficult to estimate).
- (2) The time of antitoxin administration.
- (3) Degree and extent of rigidity.
- (4) Possibility of taking food.
- (5) State of body temperature.

Another point must not be omitted, viz., the relatively frequent occurrence of mixed infections due to pyogenic organisms. Purulent inflammation of the umbilical vessels and peritonitis are frequently found in autopsies of tetanus cases. Such complications, of course, make the prognosis considerably worse.

That even very severe cases have recovered may be illustrated by the Vienna Children's Hospital:

Tetanus neonatorum, refusal of food and trismus on the eighth

day. On being lifted on the twelfth day severe general tonic spasms. On fifth and sixth day of disease, injections with 30 c.cm. tetanus serum. In spite of this, increase of symptoms. High intermittent fever (107° F.). Almost impossible to give food with a catheter so that the child nearly starved for fourteen days. Fluid given subcutaneously and by enemata. The weight of the body sunk from $2\frac{1}{2}$ lb. to 6 lb. The symptoms only began to diminish in intensity on the twenty-first day, when the child, almost reduced to a skeleton, began to take the breast. Improvement then progressed rapidly. In the ensuing months the child developed a perfectly normal, healthy appearance.

Treatment.—The results of serum therapy with tetanus nosotomum have not corresponded to the original expectations. In view of the very varied course of the disease, the results up to date are hard to judge; but the impression has not been given that since the introduction of serum therapy there has been any change in the mortality. This is not to be wondered at, because the injection of serum is only given when the typical symptoms have already appeared, that is to say, when the poison has already settled in the body cells, and can no longer be counteracted by the toxin. Serum therapy is only fully justified as a prophylactic measure. Nevertheless, the effort should be made in every case to influence the poison by incorporation of the tetanus antitoxin as early as possible; the toxin circulating in the blood may surely still be removed by injections of serum. Behring's antitoxin (250 units) is best injected, partly subcutaneously, somewhere in the umbilical region, and partly intradurally after lumbar puncture. If no effort is noticeable after the first day, the injection may be repeated.

Among other methods of treatment, may be mentioned the formalin injections recommended by Szalárdi ($\frac{1}{2}$ drop in 10 c.cm. physiological salt solution) and Bircelli's method of carbolic acid injection. The latter should be undertaken in the following way: 3 per cent. carbolic solution in sterile oil injected daily in doses of 1 c.cm. and a total of 4 to 10 c.cm., gradually increased. The injections may be continued for thirty to forty days (Fedele, Miserocehi).

Of special importance is the symptomatic treatment, which is directed towards the mitigation of the spasms which are accompanied by a great waste of tissue. The most suitable remedy is chloral hydrate, which is administered by enema, in single doses of about 0.5 gm., three to four times daily, in a 1 per cent. solution. The chloral may be administered daily for some time, which is not possible with other narcotics, as, e.g., with the momentarily effectual inhalations of chloroform. The administration of sedatives per os (bromides, calcium or chloral³) meets with difficulties, the same as feeding. Great care must be used with the latter. Only in mild cases will the child take from the breast or the bottle,

³ See pages 217, 463.

and as a rule feeding with a spoon is very unsatisfactory. In such cases food must be given with a stomach tube, which owing to the trismus must be passed through the nose. Tube feeding sometimes meets with obstruction and does not fulfil its purpose, owing to vomiting. The effort must then be made to assure to some extent the necessary fluid by subcutaneous infusions and injections into the bowel. Nutrient enemata with mother's milk may also be tried.

Erysipelas.

With the discussion of erysipelas, a disease caused by streptococci, we enter the sphere of septic diseases. On the basis of experimental researches it is assumed that the streptococcus discovered by Fehleisen is not a specific streptococcus. Erysipelas takes an important position less by its aetiology than by its localization in the skin and its clinical course. In this respect also there are transitions from the dermatitis described as erysipelas to phlegmonous and suppurating processes of the skin and subcutaneous connective tissue. The most striking factor is the marked contagiousness of the disease, and particularly the nature of the same. Although it is an infection by a streptococcus, a micro-organism which is capable of causing so many varieties of disease, erysipelas in one individual is followed, not always but very frequently, by erysipelas in another. This suggests that many strains of streptococci may easily cause erysipelatoous diseases.

The frequently described intra-uterine erysipelas infections are of particular interest. Kaltenbach, Runge and Stratz have observed, that children whose mothers developed erysipelas a few days or weeks before confinement, were born in a state of diffuse, lamellar desquamation, spread all over the body. The remarkable thing in these cases is, that the erysipelas, otherwise so dangerous to the child, should have healed in the womb. Lebedeff examined the skin of a premature child of a mother who, a few weeks ante partum, had suffered from erysipelas, and found immense numbers of cocci in the lymph spaces. Schieb reports the case of a child of a mother suffering from septic endometritis, which became ill with high fever on the first day, and on the fourth day a few hours before it died showed erysipelas in the face and head. The infection was probably due to an injury, during birth, to the palate. The case can hardly be regarded as an intra-uterine infection. It points to the most frequent source of infection for erysipelas in the newborn, viz., the lochial secretion of a woman suffering from puerperal sepsis.

Erysipelas in the newborn infant starts either at the umbilical wound, or in the cord, or in some superficial injury of the skin, which may be quite invisible. The genital organs are very frequently the starting point of the disease (fig. 88a). The lower half of the body is much more often affected than the upper half. The usual time of onset is at the end of the first week, or in the second or third week. It begins with redness and cedematous swelling in

the vicinity of the portal of infection. In typical cases these symptoms extend rapidly, in a downward direction generally, over the genitals, the thigh and buttocks, leaving the area above the level of the umbilicus quite free in most cases (Knöpfelmacher). The red and swollen portions of the skin usually feel quite warm. Erysipelas in the newborn does not always exhibit that definite limitation of the diseased area, with a sharp continuously progressing edge, which is so characteristic in the adult. The edges are sometimes rather flat and irregular; and occasionally the reddening is not very intense, but the inflammatory oedema is usually very pronounced.

Vesicles often develop on the inflamed skin, and gangrene or necrosis occurs with comparative frequency, especially on the scrotum and labia majora. These parts may at first assume a



FIG. 88a.—Erysipelas in an infant aged ten days, starting in the genitals.

circumscribed bluish-red coloration, then break down and ulcerate (Noh1). Sometimes an abscess develops, in the course of the disease, at one spot. The disease is usually fulminant in its progress, the general condition is bad; the infants are weary and exhausted, drink but little, and continually moan. The temperature may be very high or it may remain normal.

The prognosis is usually very gloomy, and most of the infants die in a few days. The post-mortem reveals merely the general signs of toxæmia or infection (degeneration of the parenchymatous organs, swelling of the spleen), or a pyæmia. Occasionally an acute purulent peritonitis is found. Achalmé constantly found streptococci in the subcutaneous tissues of newborn infants with erysipelas, but they were never inside the leucocytes, whereas in erysipelas of

adults the organisms were still in the cutis and were taken up by the leucocytes. The virulence of the disease in early infancy is perhaps explained by the absence of phagocytic activity of the leucocytes.



FIG. 551.—Same case twenty-four hours later (further down before death).

and the unhindered invasion of the streptococci in the cutis, their penetration into the lymphatic system and their flooding of the whole body (Hörigst). Nevertheless, a case of extensive erysipelas occasionally recovers (Friedjung). Purulent softening may perhaps be regarded as a favourable prognostic sign.

The contagiousness of erysipelas renders the greatest care necessary. It has repeatedly occurred that the disease has been transmitted by the hand of the doctor, midwife or nurse. It is also very dangerous for the lying-in woman. Therefore, not only in lying-in institutions, in the interest of the other newborn infants and women, is isolation of the affected infant necessary, but also in the private house the greatest care should be taken by thorough disinfection to avoid the transmission of infection. The person who is entrusted with the care of the infant should not be allowed to nurse the woman as well. During the acute stage of the disease, it is imperative that the child should be fed with drawn-off milk, instead of being applied to the breast, and even here great care is necessary.

The treatment is not very promising. As a rule it should be confined to the application of antiphlogistic compresses (lead acetate lotion, *lotio plumbi*) or ones containing 50 per cent. alcohol, $\frac{1}{4}$ to $\frac{1}{2}$ per thousand sublimate boracic solution, 1 to 2 per cent. salicylic acid, 1 to 3 per cent. peroxide of hydrogen. The compresses are best applied cold, possibly ice cold; the cold alleviates the pain. If the erysipelas can be covered on all sides by means of a bandage, as on the skull, Heubner recommends Nussbaum's *ichtyol* method (i.e., the skin is shaved, carefully disinfected, two hours later compresses of salicylic acid are applied, and then 50 per cent. *ichtyol* ointment is rubbed in for a quarter of an hour, two fingers' breadth beyond the margins of the erysipelas. The ointment is then spread the thickness of a knife blade on a piece of lint, and then fixed on by means of gauze bandages (over which an ice-bag should be applied). This procedure is repeated once or twice daily.

It may also be tried, by means of strips of sticking plaster, which are tightly applied some distance from the margin of the erysipelas, to compress the lymph spaces of the skin, thereby preventing the process spreading (Wölfler). Bier's hyperæmin has also been recommended. With regard to the general treatment and feeding, the same rules apply as in all septic diseases.

Malaria.

If a woman during pregnancy develops malaria, the disease may be transmitted to the foetus. If the placenta is intact, the malarial plasmodia probably cannot be transmitted to the foetus; therefore children of mothers with malaria may remain quite healthy. But obviously, quite insignificant lesions of the placenta are sufficient to allow of transmission. Children have often been observed who show all the signs of malarial disease shortly after birth (Peters, Ballantyne). Grandall observed in the child of a woman who had had tertian attacks before confinement, an attack of fever within the first twenty-four hours, which was repeated on the following day. Malarial plasmodia could be found in the blood.

In other cases the first attack of fever appears after some little

while, in a case observed by Cina not till the twentieth day, and in a case observed by Pies not till the thirty-second day. In spite of the period of latency, an intra-uterine transmission must, in such cases, be assumed, if, as in the last case, the confinement took place in a region free from malaria.

Children who are born with symptoms of malarial cachexia, may succumb after a few days, particularly if born prematurely; others, however, survive. Treatment with quinine may be applied successfully, even in very young infants.

CHAPTER II.

CHRONIC INFECTIOUS DISEASES.

(1) Tuberculosis.

In contrast with syphilis, which cannot by any means be regarded as a rare disease in the newly born, and presents, in the earliest period, many characteristic clinical symptoms and aspects, tuberculous diseases during the first weeks of life are extremely rare, and the clinical symptoms very indefinite, even if anatomical changes are present. In syphilis of the infant the congenital form largely prevails; congenital tuberculosis, in contrast with the acquired form, is extremely rare. The rarity of congenital tuberculosis is the cause of the number of observations on tuberculosis in early infancy being relatively small. In view of the sparsity of descriptions of the symptoms of cases observed, it is hardly possible to describe a well-defined clinical form of tuberculosis of the newly born. If the clinical aspects of tuberculosis in earliest infancy fill but a very small space in our present medical knowledge, on the other hand, just this first period of life is of paramount importance in the question of the source of infection of tuberculosis, and the hygienic and prophylactic measures to be taken.

As regards the time of infection the following possibilities exist:—

(1) Infection occurs *before* the exit of the child from the maternal genital passages (congenital tuberculosis). In such a case it may be a germinative infection; an infection *in utero* or *intra partum*. In both the latter cases the tubercle bacillus penetrates the infant's body either by the placental blood circulation or by aspiration or swallowing of the liquor amni. The child is either already born with tuberculous changes of the organs, or the foetus is even tuberculously infected at the time of birth, but shows no specific organic changes. Schlossmann calls only the first form congenital tuberculosis, and describes those cases in which merely transmission of the germs to the foetus has taken place as hereditary tuberculosis.

The expressions "congenital" and "hereditary" do not corre-

speed in this sense to the strictly logical definition of Martius, according to which everything is to be termed congenital that is already in the individual at the time of birth, and as "hereditary" only that which develops from the determinants of both sexual cells, that which is imparted to the offspring through germinal substances. But from the clinical point of view it is desirable that the children who are born with tuberculous organic changes should be differentiated from those who are not, but are born already infected. In the latter case active tuberculosis may be present, having been transmitted shortly before or during birth, and is only manifest during the course of the first weeks of life—the newborn child is in the incubation stage of tuberculosis—or it may be a case of those inactive forms of tuberculosis, not generally recognized, which are to be attributed according to Baumgarten, to the presence of latent tubercle bacilli, and remain inactive for a longer or shorter period.

(2) The infection occurs after birth (acquired tuberculosis). This may occur either by way of the respiratory tract (inhalation tuberculosis), or by way of the digestive tract (assimilation tuberculosis). Other portals of infection are of subordinate importance.

(a) Congenital (and Hereditary) Tuberculosis.

The generative infection of the ovum plays practically no part. Even though intra-ovarian infection of the latter with the tubercle bacillus assuredly occurs, every possibility of such an ovum being fertilized is, as a rule, absent; hitherto it has only been possible in birds to obtain tuberculous young from infected ova. Extra-ovarian infection of the ovum affords more favourable conditions for fertilization and further development, though this has not yet been irrefutably proved. The possibility of a generative transmission through the spermatozoa is not out of the question, but this again has not been observed irrefutably in human beings (Sitzenfrey). Without tuberculosis of the mother congenital or hereditary tuberculosis does not appear to occur.

The possibility has also been shown of the infection of the germ after fertilization. Friedmann has proved in experiments on animals that by injection of tubercle bacilli into the vagina post coitum infection is possible, as he was later able to show bacilli in the embryo. Kraemer suggests that this occurs also in human beings; he imagines that the germ, post conceptionem, can be infected by the bacilli within the uterus, either through the maternal infective material or that transmitted by the father in the semen. But this theory arouses doubt, because such quantities of tubercle bacilli never occur in the uterine cavity, as in Friedmann's experiments.

The theory of tubercle bacillus infection by the placenta stands on a firmly established basis. The normal placenta represents a filter impervious to bacteria. The supposition of a transmission

of tubercle bacilli from the maternal to the infantile blood is based on a lesion of this filter. The tubercle bacillus may be transmitted into the infantile circulation if a communication is introduced between the intervillous blood spaces and the vessels of the chorionic villi, or if the liquor amnii is infected with tubercle bacilli. The bacillus infecting the foetus may be derived either from the tuberculous placenta or from the circulating blood.

Tuberculosis of the placenta has been most carefully studied by Biedt-Hirschfeld, Schmorl and his pupils, Kockel, Geipel, Schlimper, and also by Szentmyer. The following forms may be differentiated:—

(1) Villous tubercles. These are situated either on the surface of the villi and in the intervillous spaces, or in the interior of the villi. The first appears to be the more frequent form of placental tuberculosis. The rarer primary villous tubercles with which only infantile elements are associated may exist through tubercle bacilli being forced into the villi from defects of the surrounding epithelium. The transmission of bacilli into the infant's blood takes place if a villous vessel is eroded or torn.

(2) The round cell infiltrations in the decidua basalis containing tubercle bacilli.—These may penetrate into the intervillous spaces, and from thence lead to villous tuberculosis, or render possible the transmission of tubercle bacilli into the infant's blood through rupture of the villi.

(3) Tuberculous changes in the decidua vera or in the chorionic membrana tectoria of the placenta. If these foci break through the amnion, infection of the liquor amnii may result, and lead to intestinal or pulmonary infection of the foetus.

Pronounced placental tuberculosis is only found with advanced tuberculosis of the mother. But the placenta may also harbour tubercle bacilli when no macroscopically visible tuberculous changes exist in the placenta and foetal membranes (Leutenberger). Novak and Ranzel examined ten placentae of tuberculous mothers by means of the uniform method of obtaining tubercle bacilli, and obtained positive results in seven cases. But in such cases it still remains undecided whether the bacilli are derived from the placental tissue or the maternal blood. We know that in the blood of tuberculous individuals, even in mild forms of the disease, tubercle bacilli may be present.

The transmission of bacilli from the mother to the child may occur during pregnancy, that is when the placenta is closely connected with the uterus, if the barrier is broken down between mother and child in the above manner by the tuberculous process. The possibility of such intra-uterine transmission before the onset of labour, i.e., in the foetal period, may be gathered from the fact that advanced tuberculous processes occur in children who died shortly after birth. Tuberculous disease or tubercle bacilli have been found in the foetus *in utero* of women who have died of tuberculosis during pregnancy, also in children who have been brought

into the world by Caesarean section. Transmission of tubercle bacilli to the child during birth occurs more readily and possibly more frequently than has yet been imagined if the villous vessels are torn on separation of the placenta, and in this way the transmission of bacilli into the infant's blood is rendered possible, either from tuberculous foci in the placenta or from the maternal blood (Sitzenfrey, Rietschel).

According to the previous classification infection in the fetal period would usually give rise to "congenital" tuberculosis (tuberculous changes in the body of the newborn child) and infection intra-partum to "hereditary" tuberculosis (transmission of infective tubercle bacilli into the infant's circulation, which results later in tuberculous changes).

The cases on record of congenital tuberculosis have been collected in the following table. (See also Cornet, Schlüter.)

Probably also the case of Wollstein, and possibly that of Lyle, may be considered examples of congenital tuberculosis.

As regards the clinical symptoms of congenital tuberculosis, in tuberculosis transmitted intra-partum or during the last period of pregnancy, its course is clinically latent during the first period of life, and it is only manifest after two or three months. We will not deal here with the question whether there is a longer period of latency (some years) of the tubercle bacilli, already present in the body at birth. Rietschel is of the opinion that only those cases may be considered as congenital tuberculosis in which the latter is manifest during the first six months, whereas Dietrich considers a much longer stage of latency possible for congenitally transmitted bacilli. In all these cases, whether of active or inactive tuberculosis, we are dealing with latent forms, and therefore, from a clinical point of view, with diseases of the *new-born*.

If at the moment of birth tuberculous changes in the body are present, the disease appears in a living child to lead to death in the majority of cases, either a few hours after birth or in the course of the first few weeks. As already mentioned, we have no precise observations regarding the clinical condition of such children during the first days of life. We are not sufficiently informed about the course of the temperature and weight, nor about the physical symptoms. In accordance with the source of infection, disease of the thoracic lymph glands, which results, in acquired tuberculosis, in characteristic symptoms (expiratory stenoic type of respiration, noisy cough, &c.), is not prominent in the clinical picture. The characteristic condition for the anatomical diagnosis of congenital tuberculosis is the involvement of the abdominal glands, particularly those lymphatic glands situated in the porta of the liver, which cannot always be manifested clinically in the body by characteristic symptoms (collateral circulation). If the tuberculosis has spread further, swelling of liver and spleen, general glandular swelling, also pulmonary symptoms (rhonchi and signs of consolidation), and skin tuberculosis may render the diagnosis possible,

| Author | Mother | Child | Post-mortem finding |
|----------------------------|--|--|--|
| Andrews | — | B. shortly p. p. | Tracheal and bronchial glands swollen and caseous, milary tubercles found. |
| Barni | Tuberculosis | B. 3 days p. p. | Two cavities filled with caseous material in the right lower lobe. |
| Brinley | — | B. 12 days p. p. | Numerous tubercles in liver and lungs; bacilli found. |
| Bugge | The. puer., d. after delivery | Premature (8 months), d. 30 hours p. p. | Bacilli in umbilical blood and liver vessels. |
| Dierich | Miliary the., d. 3 days after delivery; placenta without the change; bacilli found by means of anitoxin method | 2½ lb., separated immediately from mother, artificially fed, absent in the m. urium (the.) after the 9th day. In the 2nd month, moderate fever, in the 3rd month, five riles, symptoms of tuberculous d. 24 days p. p. | Caseous area in right apex; numerous tubercles in both lungs (bronchial glands neither softened nor caseous). Inosistal ulcers, small nodules in the liver. Gland the size of a hazel nut, completely caseous in parts of liver. |
| Hassinger | The. puer., d. 5 days after delivery | D. 7½ weeks p. p. | General tuberculous-miliary the., chronic the. and caseation of the glands in parts of liver. |
| Herr | B. from advanced the.; placenta contained bacilli | Fetus, 6th month | Tubercle bacilli in the liver. |
| Hochmayer | The. (father syphilitic) | Sickly and pale from birth, much coughing; in 2nd week, syph. eruption, 1 h. and infiltration, d. 30 days p. p. | Internal organs throughout tuberculosis, especially the liver. |
| Hood | — | D. 15 days | Numerous areas in liver and spleen, bacilli found. |
| Jacobi | The., died 3 weeks after delivery | 7 months fetus | Numerous milary tubercles on surface of liver, spleen, peritoneum, placenta. No bacilli found. |
| Lahmann | The., d. 3 days after delivery | D. 24 hours p. p. | Caseous the. in the glands in parts of liver and in the mesentery. Liver and spleen filled with small areas. |
| Levins | — | D. 18 days p. p. | Tubercle in lung; caseation of bronchial and tracheal glands; extensive ulcers in intestine. |
| Rudolf | Flood the. during pregnancy | D. 8 days p. p. | Large caseous nodules in liver; caseous pneumonia. |
| Sabouraud | The. puer. | D. 11 days p. p. | Liver and spleen filled with caseating tubercles (bacilli found). |
| Schoof and Bach-Hirschfeld | Miliary the., d. immediately after delivery in the 7th month of pregnancy; bacilli in the placenta | Dead fetus, Caesarian section to give a western. | Bacilli in liver, spleen, and kidney, but no the. changes. |

| Autopsy | Mother | Child | Post-mortem finding |
|--------------------|---|---|--|
| Schmitt and Kockel | Prim. tbc., d. a few days after abortion; placental tubercles | Dead fetus... | Excess in hepatic vessels. |
| Schmitt and Kockel | Prim. tbc., d. 4 days after delivery; caisson endometritis | D. 12 days p. p. — | Suprarenal capsules almost completely created. |
| Schmitt and Kockel | Miliary tbc., d. when pregnancy at 7-8th month; placenta tuberculous | Dead fetus... | Bacilli in liver, and lymphatics of hepato-duodenal ligament. |
| Steenfry | Prim. tbc., d. 1 month after delivery; placental tubercles | 8 lb. 4 oz. Fresh-looking child; separated from mother immediately p. p. At first good increase in weight (fed by wet nurse). Some cough. In 3rd month more severe cough, increase of temp. in evening, later riles and consolidation. The. bar. in sputum. D. 5 months | Chronic tbc. of lung lymphatic glands. Intestinal tbc. |
| Steenfry | D. 5 days after delivery; plac. tbc. | Small, thin. Separated from mother immediately after birth. No increase in weight. Finally decrease. Fever. D. 6 weeks p. p. | Chronic tbc. of lymphatic glands (usual parts of liver), lungs, liver, spleen, thyroid gland. |
| Steenfry | Chron. general tbc. d. 4 months after delivery. Isolated tubercle in plac. | Artificial prem. birth. 4 lb. (fed by wet nurse). 1st month decrease in weight, then increase. In 3rd month symptoms of riles and consolidation. D. 3 months (4 lb.) | Chronic tbc. of lungs. Intestinal ulcers. Tbc. of mesenteric lymphatic glands. Miliary tbc. (liver, spleen, kidney). |
| Strass | Parents specified healthy (?) | D. 3 weeks p. p. — | Cavating tubercles of bronch., and mesent. glands, liver, spleen, lungs. Large tubercle in liver. |
| Vesegreny | Miliary tbc. | Rise in temp. from birth. D. 31 days p. p. | Tbc. of glands in the porta of liver. Miliary tbc. |
| Tarl | D. from prim. tbc. 3 months after delivery. Flaccid without naked eye changes | (Observed from 10th day.) At first subfebrile temp., with 4 weeks intermittent fever up to 102°. Emaciation since 12th day. Enlargement of liver and spleen. Veins obvious on chest and abdomen. Increase of abd. circumference. Swelling of palpable lymphatic glands. Symptoms of bronch. in 6th week. Pirquet's reaction on 17th day pos. D. 7 weeks | General advanced tbc. in lymphatic and blood vessels. Severe lesions in portal lymphatic glands, which form a mass about 5 cm. in size, with firm softening. Large splenic tumour with numerous tubercles. The thoracic lymphatic glands show much slighter changes. |

apart from the invariably advancing decline in such cases (Zarfl). In this stage the X-ray findings may also be of use.

Merbel's case of congenital tuberculosis of bone (lesions in the palate and trochanter) is quite unique. In Dietrich's case there was on the tenth day an abscess in the os sacrum, the tuberculous nature of which was doubtful. Coenot mentions three cases of surgical tuberculosis reported by Lannelongue: osteo-arthritis of the knee (appeared fifteen days p.p.); tuberculous abscesses in the malleolar region (in a child of three weeks); tuberculous osteitis (in a child of ten days).

Also concerning the condition of the tuberculin reaction and its diagnostic value, no observations are available in tuberculosis of the newly born. In consideration of the extreme rarity of the disease, it is not surprising that the tuberculin reaction in the newly born is generally negative (Schreiber, Behrend, Calmette, Duverger, Petruseliky, Polen and Griemert, Bondy, Longo). Engel and Bauer report a positive cutaneous reaction in a healthy breast-fed child that did not react to tuberculin injection; Brückner found in a child of fifteen days a positive Pirquet reaction without being able to find macroscopically tuberculosis at the post-mortem. Apart from these doubtless non-specific cutaneous reactions, the youngest ascertained tuberculous child, in which Pirquet's reaction was positive, was Zarfl's case of seventeen days. Rietschel's case of hereditary tuberculosis was repeatedly negative to Pirquet's test. A case observed by Ibrahim of a child infected during the first five weeks was negative in the seventh week, and at the next inoculation at the age of three months was positive. Ibrahim declares it to be uncertain whether the inability to react, owing to its early age, was responsible for the negative result, or whether the child observed by him at the time of the first inoculation was on the point of becoming allergic. The latter he considers the most probable. At present we can merely say that the child infected with tuberculosis, but not yet tuberculous, probably always has a negative reaction without our knowing at what time the tuberculin reaction begins to be positive. Concerning the capability of reaction of the newborn child, who is already tuberculous within the first two weeks of life, our information is still lacking. Up to date no tuberculous newborn child has been tested with tuberculin. Only systematic examination of children of tuberculous mothers will give the information required.

(b) Acquired Tuberculosis.

Even in pronounced tuberculosis of the mother, with actual tuberculosis of the placenta and umbilical cord, the child may escape the disease (Sitzentrey). Experiments on animals by Bossi and Pantow have shown that in infection of pregnant animals with tubercle bacilli the foetus in the majority of cases remains free from tuberculosis. It is also a fact that children of tuberculous mothers, if immediately after birth they are removed from the

mother and the tuberculous environment, do not contract tuberculosis. It is only very tuberculous women who bear tuberculous children; they often die during or shortly after confinement from miliary tuberculosis. In a large number of tuberculous pregnant women abortion results, or, in view of the deleterious effect of pregnancy with severe tuberculosis, abortion is artificially procured. In other cases pregnancy will not occur at all. Congenital tuberculosis, for these reasons, is an extremely rare occurrence. Opinions are divided concerning the part played by tubercle bacilli in the foetus, with moderate or mild tuberculosis of the mother. The majority of clinicians and pathological anatomists are of opinion that, in contrast to hereditary tuberculosis of the child, which is due to the presence of latent bacilli, and only breaks out after a more or less long period of latency, tuberculosis acquired post partum plays by far the most predominant part.

The clinical symptoms of acquired tuberculosis only become manifest when the newborn period is over. Although the clinical study of acquired tuberculosis is outside the sphere of our own observations, the prophylaxis of tuberculosis plays all the more important part in the very first period of life. We know at least that tuberculosis is all the more dangerous the earlier in life the infection occurs.

Recent researches concerning the sources of infection of tuberculosis have proved that by far the most frequent and important is the aerogenous. It was proved by careful anatomical examinations of tuberculous infants, on the part of Kuss, Albrecht and Ghon, that the first localization of tuberculosis is generally a primary pulmonary focus, from which secondary affections of bronchial glands result. This type of infection is far more significant for human beings than the intestinal type. It follows, therefore, that the first and most important object is to protect the newborn child from aerogenous infection.

The following experiment of H. Reich, communicated by Range, is proof of the importance of aerogenous infection. A tuberculous midwife made it a practice of trying to resuscitate children with asphyxia by aspirating the mucus with her own mouth or by breathing into their mouths. In the course of thirteen months ten of these children died from tuberculous meningitis, whereas in the practice of other midwives in the same place not a single child contracted the disease.

If in the immediate neighbourhood of the child there is anyone with open tuberculosis, either mother or father, nurse or anybody else, remaining in the same atmosphere as the child, then the latter should be removed as quickly as possible from its tuberculous environment. The sooner the child is brought into surroundings free from tuberculosis, the greater are its chances of remaining healthy. It has repeatedly been observed that children who have been separated from tuberculous mothers immediately after birth have escaped the infection. Bernheim's observation may serve as an example. Of three tuberculous mothers with twins, one of each pair was taken to the country and artificially fed, the other being

fed by a wet-nurse in the mother's house; the three isolated children remained healthy, the three others remaining in the respective parents' house died, in spite of feeding by a wet-nurse. In what a short time infection may result is shown by the following case. A child was brought to the Vienna hospital for observation that had only been three hours with its tuberculous mother and then removed. Yet the child died after three months, and the post-mortem made by Gross showed general extensive milary tuberculosis, which had to be regarded as acquired tuberculosis owing to the slight changes in the portal lymphatic glands and the severe changes of the bronchial lymphatic glands on one side. It is not out of the question that this was a case of aspiration of liquor amnii containing tubercle bacilli, but it was not probable owing to the dominance of the bronchial gland affection of one side and of the relatively long duration of life. Georgi also considered a case as acquired tuberculosis in a child of a healthy mother which died on its fourth day.

An unusually evident example of aerogenous tuberculosis acquired during the first period of life is the following case of Zarß's: A nineteen-day-old child of an extremely tuberculous mother, who during its first week of life was in the tuberculosis section of a hospital, developed lobular pneumonia, from which it died after six days. The anatomico-histological examination showed the presence of a pulmonary focus full of tubercle bacilli in an upper lobe in addition to the parts of the lung affected by pneumonia, whereas the lymphatic glands, like the rest of the body, were found to be entirely free from tuberculous changes.

While the enormous danger of a tuberculous atmosphere for a child is undoubted, yet opinions are divided regarding the danger of the milk of a tuberculous mother. Efforts have been made to apply to human beings the experiments made on animals. We know that in udder tuberculosis of the cow tubercle bacilli can be transmitted in the milk (though in the cow, even in generalized tuberculosis, the milk may remain free from bacilli). But mammary tuberculosis is so uncommon in human beings that, in the question of the possibility of suckling, it practically plays no part. With regard to the occurrence of tubercle bacilli in the milk in the ordinary localization of human tuberculosis the results do not always agree. Whereas Schlossmann found negative results in four, Rubinowitsch in three, Fesser and Behler in seven cases with inoculation, Roger-Garnier and Escherich each found positive results in one, and Nosenwitsch in three cases. Particularly worthy of note are the results recently published by Kurashige, Mayeyama, and Yamada; in pulmonary tuberculosis of the mother they were able to find tubercle bacilli in the mother's milk:—

In the prodromal and 1st stage among 13 cases, 10 times.

| | | | | | | | |
|---|---|-----|---|---|---|---|---|
| " | " | 2nd | " | " | 5 | " | 5 |
| " | " | 3rd | " | " | 2 | " | 2 |

The significance of intestinal infection, to which Behring attached

so much importance, and which also found many adherents among pediatricists, must take a subordinate position owing to the anatomically established fact that inhalation tuberculosis predominates considerably over that conveyed through food. In view, however, of the well-known permeability of the intestinal mucous membrane of the newly-born for bacteria, the possibility of intestinal infection can by no means be denied. The statistics of Deutsch led to a most remarkable result: of sixteen children of actively tuberculous mothers who suckled, five developed tuberculosis (two of them died), whereas four children of actively tuberculous mothers who did not suckle, were not infected, although they remained with their mothers. It is certainly not proved that these were cases of intestinal infection.

As a rule, in obvious pulmonary tuberculosis of the mother, suckling should under all circumstances be prohibited, and the same should be recommended with every active tuberculosis of the mother. In those cases the primary object in forbidding suckling is the removal of the child from the coughing mother, or rather from the atmosphere containing bacilli, and is done more on account of the danger of tuberculosis from inhalation than from possible transmission of bacilli through the mother's milk; though pronounced tuberculosis of the mother should, under all circumstances, be considered as a contra-indication to suckling—in the interest of both parties. It is the duty of the doctor, in such cases, to do his utmost to procure the removal of the child from its mother's vicinity, at any rate for the first few months of life.

Also with those mothers, known to be tuberculous, but who show no manifest signs at the time of confinement and lactation, much precaution is necessary. If the removal of the child from the mother should meet with insuperable difficulties and the child must remain in the parents' house, then according to our clinical experience, it is wiser not to subject the child to further danger by artificial feeding, but to let it feed at the breast. If neither clinical nor physical tuberculous changes can be found in the mother, if the condition of sputum is negative, if the tuberculosis is not only latent and inactive and confined possibly to a history of catarrh at the apex of the lung, then, if the child remains with its mother, suckling at the breast should not only be permitted but advised. It has been admitted both by pediatricists (Schlossmann) and gynecologists (Fischer) that in such cases suckling has also no detrimental consequences for the mother. If a wet-nurse can be procured, in doubtful cases this is the best expedient.

Apart from aerogenous and intestinal portals of infection others are of subordinate importance. Occasionally infections with tuberculosis may be derived from the external skin: e.g., in ritual circumcision transmission of tuberculosis has been observed (J. Neumann). Infection probably resulted from the common practice of sucking the wound with the mouth on the part of the circumciser, in order to stop the bleeding; the disease manifested itself a few

weeks later by the appearance of swelling and ulcers accompanied with tuberculosis of the inguinal glands. Epstein reports on similar forms of inoculation tuberculosis connected with piercing of the ear lobes. His observations, which apply to the period of early infancy, furnish weighty evidence against the danger of this barbaric custom, which is unfortunately still so popular.

(c) **The Influence of the Mother's Tuberculosis on the Child, without Tuberculous Disease of the Latter.**

PARATUBERCULOSIS.

Though children of tuberculous mothers can be born entirely free from tuberculosis, and with suitable prophylaxis may be permanently preserved from it (Weinberg), it is nevertheless remarkable that a relatively high mortality is produced by it; this applies in particular to premature children. It has been observed in a number of cases, that children of tuberculous women, that died shortly after birth or during the first few days, showed no tuberculous changes at the post-mortem. In a few of these cases bacterial examination showed the presence of tubercle bacilli in the infant's organs or in the umbilical blood (Schmorl, Birch-Hirschfeld, Bugge, Schrampt, Lönke and Thiercelin); therefore the children, if they had survived, would have been attacked later with tuberculosis, though they did not die of it. Apart from the afore-mentioned experiments on animals, several cases in human pathology have been known, in which not only the microscopical post-mortem result was negative, in respect of tuberculosis, but also the bacterial examination of the organs, and their inoculation into animals, gave a completely negative result (Schmorl and Kockel, Sitzenfrey, Beneke and Kürbitz). In such cases one is justified in presuming injury to the fetus through poisonous products of the metabolism of tubercle bacilli, through a more or less virulent toxin transmitted to the infant's body by the placenta. If the children continue to live and escape tuberculosis, they may still present a strikingly poor development, constitutional weakness and lack of vitality. Schlossmann communicates the following case in point: the child of a mother suffering from chronic tuberculosis continued to lose weight, in spite of the most favourable nursing conditions, and died suddenly after seven weeks; the post-mortem showed entirely normal organs, only slightly atrophied.

In statistics regarding the fate of premature children Planck and Devin found a higher morbidity and mortality in children of tuberculous mothers; the children also showed otherwise poor increase in weight and markedly poor development.

Schlossmann describes these signs of constitutional weakness from tuberculosis of the parents as paratuberculous symptoms. Possibly this has a bearing on the question of "disposition" to tuberculosis in individuals with an hereditary taint.

In respect of its mode of origin, congenital leprosy appears to be similar to congenital tuberculosis. Sugai found lepra bacilli in the blood of the umbilical vessels in a newborn child and a fetus, both belonging to a leprosy family. A leprosy affection could not be shown in the body of the newly born (a condition which has similarly often been observed with tuberculosis infections). The placenta showed typical leprosy changes.

(2) Syphilis.

Whereas in the earliest period of infancy, tuberculosis in the great majority of cases is acquired post partum and is only rarely inherited or congenital, the latter is the rule with syphilis. Acquired syphilis does occasionally occur in early infancy, but it plays a subordinate part.

The mode of infection of inherited syphilis has for long been a subject of lively scientific controversy, and in spite of the discovery of the syphilitic organism and the serum diagnosis, it cannot be regarded as finally elucidated. As in tuberculosis, there are two possibilities in syphilis to be distinguished, the generative and post-conceptual placental transmission. Whereas in congenital tuberculosis the mother is invariably diseased, and for purely clinical reasons the question as to the possibility of any other than the placental transmission is not of essential importance, in syphilis the frequent absence in the mother of any symptoms of the disease renders the question of generative transmission of principal clinical interest. The *Spirochaeta pallida* has been found in the human ovary and ovum (Wolfer, Hoffmann and Levaditi) and thereby the possibility of syphilis from infection of an ovum has been proved. But whether such ova are capable of development, and chiefly whether they can sustain development till the child is viable, is just as doubtful as in tuberculosis. In practice it is not a very important question whether syphilis transmitted by the mother has already existed in the ovum, or was transmitted per placentam during the foetal period. Of far greater importance is the question as to the possibility of infection of the ovum by the semen of the father, without the mother being diseased. Finger and Landsteiner have proved that the semen of syphilitic subjects may be virulent. The questions therefore arise, whether this virus can be passed into the foetus, proliferate and cause syphilitic disease, and also with such a type of infection whether the mother can remain healthy. As a rule, the mother of a syphilitic child, who is herself clinically free of syphilis, is immune to the disease of the child. This "Colles-Baumes immunity" is said to be permanent, and according to the opinion of those who reject the theory of inherited syphilis of the child without the mother being diseased (Matzner), it must be classified as an active immunity, which can only be acquired through an attack of the disease, even if the latter has pursued an entirely latent course. Those authorities who

consider possible the purely paternal transmission of hereditary syphilis without the mother being diseased (Finger, Hochsinger) support their theories on the clinical fact that such women, in spite of years of observation, do not ever show even the slightest signs of syphilis, and that Colles' immunity is possibly often only apparently permanent, because no further opportunity for infection has been given, and that there may also be exceptions to Colles' law, viz., infection of a healthy mother through her own syphilitic child. Finger explains the immunity itself thus: he believes there to be intra-peritoneal inoculation with syphilis virus at the time of sexual intercourse, a mode of transmission which, e.g., with rabies, does not lead to infection but immunity. The weightiest objection to the occurrence of purely paternal syphilis lies in the fact that just those children whose mothers are clinically free from syphilis and with whom accordingly a paternal infection must be taken for granted, show a relatively benign course of the disease and frequently show symptoms of the disease, not immediately after birth but several weeks later. Just in those cases where the infection takes place at such an early period should one expect (as Rietschel rightly observes) a particularly severe course of syphilis, for it is hard to explain how the spirochaetes in such cases could remain latent for nine months in the infant's body. Finger's explanation of this extraordinary condition is, that the spirochaete have the property of remaining a long time in a state of inaction, so a certain extent encapsuled, that with germinative transmission virus is only transmitted once and then not for a long period as with placental transmission, and finally that the child, in purely paternal syphilis, thrives in the uterus of a healthy mother.

Wassermann's reaction has recently been employed in the decision of this question, which is of so much practical importance. Knöpfelmacher and Leindorff found the reaction positive in about 57 per cent. of healthy mothers of heredo-syphilitic children, i.e., about the same percentage as in syphilitic subjects in the latent stage. According to Reuben's statistics, Wassermann's reaction is positive in 72 per cent. of mothers free of symptoms; J. Bauer and Rietschel obtained nearly 100 per cent. positive reactions in mothers whom they examined. Hochsinger explains this condition by the transmission of immunizing substances from the foetus infected by its father to the mother during pregnancy, so that in this way also no decision can be made regarding the occurrence of a germinative transmission of syphilis.

If opinions are divided as to the possibility of germinative transmission of syphilis and also as to the frequency (if admitted) of such a mode of infection, then the possibility of a placental transmission must also be admitted by adherents of the germinative theory.

The placenta may be syphilitically diseased in its foetal or maternal portion, so that, as in tuberculosis, transmission of germs may take place from the maternal to the infant's blood; but accord-

ing to the researches of Nattan-Larrier, for the transmission of sporilla, an anatomically proved lesion of the placenta does not appear to be necessary. At any rate, in the case of relapsing fever sporilla it was found that they could pass through the ectodermal elements of the placenta and the endothelial cells of the fetal capillaries. Researches hitherto, concerning the presence of the *Spirochaeta pallida* in the placenta of syphilitic women, have all led to the conclusion that its occurrence is uncommon and slight. In the placentas of syphilitic fetuses spirochaetes have not been found in all cases; according to Grafenberg only in about 40 per cent. If spirochaetes are present, they are to be found in the mucous membrane of the villi, round the villus capillaries, in the periplasm of the superficial epithelium and between the latter and finally in the wall of the placental branches of the umbilical vessels and in the walls of the umbilical vessels (Schoernheim). Bab believes that the placenta possesses the property of destroying spirochaetes; he attributes to it the rôle of a protective barrier against the passage and neutralization of poisonous substances, "like a large umbilical lymphatic gland interposed between mother and child." But the protection is relatively a limited one and cannot prevent permanently the malignant progress of the disease.

If the possibility is present of the passage of spirochaetes through the placenta, at any time during pregnancy, then great significance must be attached probably to that period of birth when the connection between placenta and uterus is severed and ruptures of the vessels occur. Rietschel in particular considers this mode of infection intrapartum as very frequent and explains the fact logically, that the majority of syphilitic children only show symptoms of the disease after a certain stage of latency or incubation of several weeks. According to Rietschel's theory the children are born infected, though healthy, a condition which we are justified in assuming in many cases of congenital tuberculosis. An important support for this theory is the symptom, frequently observed, that children of a syphilitic mother at first react negatively to Wassermann's test and later positively, after the outbreak of syphilitic symptoms (Halberstaedter, E. Müller and Reider, Rietschel, Reuben).

Clinical Symptoms of Syphilis in the Newborn.

It is well known that syphilis is one of the most frequent causes of intra-uterine death of the fetus. The fetus becomes macerated and is often only after two or three weeks expelled in a decomposed condition. The death of the fetus is either a result of severe syphilitic organic changes or of poisoning by the syphilitic virus. Also severe placental disease and consequent disturbance of circulation, may result in the death of the fetus, especially during the first period of pregnancy. Syphilis often gives rise to premature birth.

Whether the children are born prematurely or at the normal

termination of pregnancy, they do not necessarily show symptoms of the disease; indeed, it may be maintained that in the majority of cases syphilis is only manifest after a period of latency, generally between the fourth and eighth week. Even on the closest examination the children often appear to be completely normal at first, and one may assume that in such cases the internal organs also do not show any anatomical syphilitic changes. In other cases, before syphilis is manifest, slight indefinite symptoms exist, possibly oozza, or slight enlargement of the spleen. Such cases lead up to actual syphilis of the newborn.

Premature, as well as full-time children, sometimes exhibit a remarkably marasmic appearance, without any of the specific cutaneous changes existing. The skin is wrinkled and limp, the facial expression senile—an aspect that one usually sees only in older, very unhealthy infants in an extreme stage of malnutrition. Such children frequently die shortly after birth, and the post-mortem shows severe syphilitic changes in the internal organs. This is the type of true visceral syphilis, such as may be observed in abortions. Fetal syphilis which attacks chiefly the internal organs is prolonged also into the extra-uterine period.

Specific changes in the internal organs are found in most children who are born with syphilitic skin changes. This is a peculiarity of syphilis, which appears during the newborn period, that explains its much more severe course and worse prognosis as compared with the forms which develop later.

Nevertheless the fact must be emphasized that also these forms, which appear during the first week, do not always occur in children on whom the external stamp of visceral syphilis is printed. Though they are often under weight, thin, sickly looking children, yet not infrequently syphilitic newborn children come under observation whose appearance immediately after birth causes no suspicion of the disease. Even if the eruption appears, the clinical picture may be similar to that of an older syphilitic infant, that need not necessarily be in any way marasmic, but often has the general appearance of a healthy breast-fed child. In such cases the visceral changes are slight. It is important to draw attention to this, as it is a common error to think that syphilis manifested post partum must always be combined with a marasmic appearance.

Whereas in children with severe general visceral syphilis the organism is flooded with spirochaetes, so that one is justified in speaking of spirochaetal septicaemia, in other cases the anatomical changes and presence of spirochaetes are confined to single organs or systems. Probably the quantity of infective material, the time of intra-uterine infection, possibly also the transmission of immunizing substances from the mother to the foetus, play an important part here. Also it may occur in the very first period of life, that syphilis is confined merely to changes of the skin and osseous system, and of a parenchymatous organ, &c. It happens therefore that the clinical aspect of syphilis is extremely multiform in the newly born,

that from severe generalized syphilis all grades, even to *asymptomatic* forms, occur, and the most various kinds of combinations are possible. The diagnosis of congenital syphilis can sometimes be formed at the first glance, but sometimes affords the greatest difficulties even to the highly skilled.

We will now discuss the changes in the various organs caused by syphilis, that take place in the newly born.

Skin.

It is possible to differentiate between diffuse and circumscribed



FIG. 52.—*Pompholyx syphilitica plantaris*.

skin changes. The so-called diffuse superficial syphilis (Hochsinger) attacks chiefly the skin of the face, the hair and the region of the mouth. The parts of the skin attacked show a peculiar, pale brownish colour. Owing to brittleness of the skin rhagades may easily develop, particularly on the lips, nose and eyelids. The second place of predilection for diffuse skin infiltration are the palms of the hands and soles of the feet, which assume a coarse, shiny appearance, sometimes reddish or blue-reddish, sometimes more copper-coloured or brownish. The surface is either smooth or shows cracks in the upper horny layers of the epidermis, which sometimes detaches itself in large lamellous scales. The diffuse

superficial syphilide is according to Hochsinger's experience never congenital, but is frequently the herald of the cutaneous manifestations of syphilis. During the first period of life the brownish sun, that lends such a characteristic stamp to the skin infiltration, is often absent. The skin is either bluish-red or shows a faded yellow colour, which is derived from the co-existing jaundice. A certain amount of caution must be exercised in judging these shades of colour; for one often finds this blue-red colour of the hands and feet, also a certain shiny appearance of the skin, in quite healthy children during the first few days, especially with a subnormal temperature.

Of circumscribed skin changes, pemphigus syphiliticus must be mentioned first of all, a syphilitic exanthem which is relatively often congenital, or is manifested during the first few days. It consists of vesicles, varying in size from that of a lentil to a farthing, the contents of which are opaque with pus, the base being formed by a brownish-red infiltrated area and the vesicles are surrounded with a margin of infiltration. In rare cases the vesicle contents may be haemorrhagic. Sometimes they may be not definitely purulent, but merely opaque like milk, though never serous. The vesicles may be confluent, so that on rupture of the often pulpy tops of the vesicles large irregular weeping surfaces may be formed, surrounded by shreds of epidermis. The pemphigus efflorescences are often situated on the palms of the hands and soles of the feet (fig. 82), occasionally only on the latter, but often on other parts of the body as well. But the palms and soles need not always be attacked; the conclusion must not be drawn from the absence of lesions in these situations that the pemphigus in question is not of a syphilitic nature. Typical pemphigus neonatorum is an ominous symptom; it is generally found in severe cases which end fatally.

Besides pemphigus there are to be found even in the newborn child maculo-papular exanthemata in the form of superficial efflorescences, from the size of a hempseed to that of a lentil, of red or purple colour, which are fairly sharply defined, but often not very easy to distinguish from the hyperæmic skin of the newborn. A favourite spot for these efflorescences is also the soles of feet and palms of hands, as well as other parts of the extremities, the trunk and face. Their flat, roundish and well circumscribed form, also a certain characteristic shiny appearance, make the diagnosis possible without much difficulty; but the brownish infiltration, otherwise so characteristic of the syphilitic papules, and which is visible after subsidence of the hyperæmia, is often absent in fresh eruptions (Brandweiner). Occasionally in the centre of a large efflorescence a slight grey elevation of the epidermis is noticed, and ultimately true formation of vesicles, &c. In this way there arise papulo-vesicular or papulo-pustular rashes (fig. 90); the vesicles are usually smaller than in the case of malignant pemphigus, referred to previously, their contents are of milky turbidity and rarely definitely yellow, their enveloping membrane is delicate and rarely tense, usually rather soft and frequently adherent to the base of the

vesicle in a few places. These varieties of rashes are generally found in the milder cases.



FIG. 50.—Papule pustule syphilitica (infant five days old).

As far as the external aspect is concerned two definite types are distinguishable. (1) Infants under weight, thin and wasted, with severe skin lesions and infiltrations in the characteristic situations

and widespread syphilitic pemphigus, suffering from marked rhinitis and easily recognizable enlargement of liver and spleen. (2) Infants who look well at birth, with rosy smooth skin, but in whom mucito-papular or vesicular lesions appear within a few days, enabling the diagnosis of syphilis to be made, which could previously only be suspected from the presence of a slight coryza or a little enlargement of the spleen.

Mucous Membranes.

Syphilitic rhinitis is the most frequent of the affections of the mucous membranes. It is one of the most important early symptoms of syphilis and is very seldom absent in syphilis of the newborn. Hochsinger collected sixty-five cases, of which thirty-five infants were born with rhinitis or manifested it very soon after birth; in five it appeared within the first week, in four it appeared in the second week, and in another four in the third week, while in two it was delayed until the fourth week. Syphilitic rhinitis is due to a hypertrophic inflammation of the nasal mucous membrane and great swelling of the nasal cartilages—a condition which often starts during intra-uterine life. The nasal space becomes narrowed, so that the characteristic snuffling respiration is caused. A mucopurulent and often a blood-stained discharge may become super-added to this dry coryza, and the rattling of the secretion will then be heard in addition to the snuffles. The nostrils will be covered with recent discharge, crusts and scabs.

The mucous membranes of the mouth, conjunctiva and vulva do not usually present any specific changes in the newborn.

Superficial Lymphatic Glands.

The clinical picture of congenital syphilis does not usually include multiple swellings of the superficial lymphatic glands; certainly they do not frequently undergo any significant enlargement during intra-uterine life. Enlargement of the glands behind the elbow, which is very characteristic of syphilis, is sometimes present in the earliest days of life, especially if there be any osteochondritis of the lower end of the humerus; but these glands are very small (of the size of a hemp seed to a pea) and are easily overlooked. In all suspicious cases the internal brachial groove must be carefully palpated. There is no disease besides syphilis in which it is possible to feel the glands behind the elbow at such an early age.

Umbilicus and Umbilical Cord.

A syphilitic umbilical cord often shows characteristic vascular disease, being thickened and tough. Reports vary in regard to the presence of spirœchetes in the cord. Graefenberg found the spirœchetes in most cases, but Böh only found them in 9 per cent. According to Graefenberg, the spirœchetes are found in special profusion near the insertion of cord into the skin. This

author attributes great importance to the finding of the spirochaetes for determining whether infection exists in the infant of syphilitic parents. He says: "If spirochaetes are found in the fetal end of the umbilical cord the infant is infected; if, however, no spirochaetes are found in an apparently healthy infant, the child must be regarded as healthy even if the parents are syphilitic."

After detachment of the remains of the umbilical cord a thickening and redness appear in many cases in the umbilical region, without acute inflammatory symptoms or fever existing. The tumour disintegrates gradually, leaving behind a deep sharp-edged dirty ulcer. These changes which, according to histological examination, are certainly of a syphilitic nature, heal quickly under anti-syphilitic treatment (Hutinel). Possibly the pigmentations of the navel, so frequently occurring in syphilitic infants, are connected with these specific kinds of processes during the healing of the umbilical wound.

The relatively frequent umbilical haemorrhages in syphilitic children are probably mostly due to diseases of the wall of the umbilical vessels, which interfere with their contraction (Hochsinger). They generally appear after detachment of the remains of the umbilical cord.

Lungs.

In fetal syphilis the lungs are very often diseased. The affection either occurs as "gumma," mostly nodules the size of a peppercorn or a pea, situated sub-pleurally, or as the infiltration of the whole lung or large sections, known as "pneumonia alba." White pneumonia is due to proliferations and cellular infiltration of the interstitial tissue, proliferations of the vascular intima and exudation into the alveoli and bronchial lumina. If the infiltration spreads over large portions of the lung, the viability of the foetus is *a priori* out of the question. Most children, if born alive, die shortly after birth. Distinct physical symptoms of infiltration in congenital syphilitic children are probably only rarely due to syphilitic pulmonary changes, but far more to secondary non-syphilitic pneumonic areas. With only slight spreading of the process syphilitic pneumonia may, however, permit of a longer duration of life (Stuhl's case, a child of seventeen days).

Thymus.

One finds in the thymus of syphilitic newborn children smooth-edged, sharply defined cavities, sometimes the size of a cherry stone, but generally smaller, and containing fluid of a purulent nature. These are epithelial proliferations which surround larger or smaller cavities, filled with leucocytes. These so-called Dubois' abscesses, which are attributed by many to arrest of development, generally contain abundant spirochaetes, whereby their syphilitic nature is established (Schridde, Simmonds, Rach). Clinical symptoms of this condition are not known.

Circulatory System.

It is essentially the small vessels which are primarily attacked by syphilis. Their involvement gives rise, on the one hand, to circulatory disturbances, formation of areas of coagulation necrosis, &c., in the various organs, including the heart and large vessels, and on the other hand to vascular rupture and hæmorrhages owing to diseases of the vessel wall. The latter condition is found, not only in areas showing pronounced inflammatory changes (hæmorrhagic rhinitis, hæmorrhagic pemphigus), but also as a manifestation of a hæmorrhagic disease (profuse nose bleeding, hæmorrhages from the navel, metrorrhagia, gastro-intestinal hæmorrhages, pulmonary hæmorrhages, hæmothorax, &c.). It is doubtful whether so-called "syphilis hæmorrhagica neonatorum" should be considered a pure form of syphilis. In such cases *papercæ* have been found in the blood and many authorities are of opinion that the hæmorrhagic form of syphilis is nothing more than a combination of syphilis with sepsis (Snodgrass, Finkelstein, Löwenberg, Hochsinger). It has not been shown that this theory holds good for all cases.

Spirochaetes have repeatedly been shown in the wall of smaller vessels, especially those embedded in the organs. The walls of large vessels (aorta, pulmonary artery) have also often been found affected (C. Bruns). According to Rach and Wiesner such affections of syphilitic children, who die during the first few days or weeks, are relatively frequently phenomena (62-67·4 per cent.), while the affection of the main vessels does not necessarily run parallel with the other syphilitic organic changes. The disease is not always merely confined to the external coat of the vessel, but may also affect the media. Cellular infiltration and proliferation may be found microscopically with obliteration of the *vaso vasorum*, but, strangely enough, there are generally no spirochaetes in the affected areas, so that the changes of the vessels are probably associated with toxic influences. Reboudi examined especially the aortas of syphilitic fetuses and newborn children, and found in the majority of cases a more or less pronounced vasculitis of the *vaso vasorum* with fresh cellular infiltration of the neighbouring tissue, the adventitia and media (mesoaortitis) and in rarer cases infiltration with new vessel formation in the connective tissue, particularly of the intima, in which it assumes a pronounced proliferating character (panaortitis with endoaortitis vegetans).

As far as we know, the changes in the large vessels in the young infant have no clinical manifestations, and are therefore more of anatomical than clinical interest. But according to Rach and Wiesner they may develop further, so that possibly many juvenile arterioscleroses may be due to congenital syphilis. It is also quite possible that milder forms of aortitis may disturb the normal development of the aorta, though clinical proof of this is absent.

Vascular affections in the region of the heart may result in formation of multiple necrotic areas in the myocardium. Changes

in the autonomic cardiac ganglia also occur, which may lead to the sudden death of syphilitic infants (Hochsinger, Winogradon).

Some authorities impute to syphilis a certain influence on the origin of congenital cardiac defects. The well known coincidence of congenital syphilis with malformations and arrest of development is probably in question and not the results of syphilitic tissue diseases.

Liver.

In fetal syphilis the liver generally shows an abundant quantity of spirochaetes. As the latter reach the infant's body through the umbilical vein, it can be understood that the liver is very frequently attacked by syphilitic disease.

The anatomical changes in the liver of syphilitic children, who die shortly after birth, are primarily due to interstitial inflammatory processes which are manifested in great proliferation and cellular infiltration of the connective tissue in the region of the vessels and between the acini. This results in diffuse enlargement and induration of the whole organ. Occasionally the so-called military gumata are found which are very small areas up to the size of a millet grain, which are formed of granulation tissue with a central necrosis or masses of detritus and leucocytes.

The changes are not always of so severe a nature. Even favourable cases often show a more or less pronounced enlargement of the liver. Hochsinger found a considerable bulging of the liver above the costal arch in the nipple line occurring in hardly 3 per cent. of non-suspected syphilitic children of the first six months, as compared with 31 per cent. occurring in syphilitic infants. The enlargement of the liver is therefore a characteristic symptom of syphilis, and in fact without any essential hardening of that organ, but this does not mean that it is invariably pathognomonic of syphilis. Not infrequently, even in pronounced syphilis, there is no obvious enlargement of the liver; on the other hand, other diseases also (such as tuberculosis) may lead to this condition.

Whereas a slight enlargement of the liver, without any essential induration, may present a favourable prognosis—with anti-syphilitic treatment the enlargement will promptly subside—a large, hard tumour of the liver, as a manifestation of severe visceral syphilis, presents a very bad prognosis. In such cases the liver may fill a large part of the abdominal cavity and distend the abdomen enormously. The skin frequently shows dilated veins. Such children are, as a rule, not viable. If severe forms of liver disease recover, a process of shrinking must be expected, though those cirrhotic processes, due to syphilis, are hardly clinically known in this period of infancy. Possibly hypertrophic, biliary cirrhoses may also develop on a syphilitic basis (see p. 271).

Isolated cases of "uterus gravis" have been causally connected with syphilis (see p. 468), but it must be emphasized that severe uterus does not belong to this form of syphilis of the liver. It

cannot even be asserted that icterus neonatorum in such cases develops to an unusual degree of intensity. Severe icterus is only to be expected in those cases in which, as the result of syphilitic disease, obliteration of the bile ducts takes place (see p. 272).

Lillemann draws attention to the occurrence of isolated congenital syphilis of the liver. He reports on three well-developed children of syphilitic mothers, that showed no external signs of syphilis and died shortly after birth. The post-mortem showed no pathological changes apart from syphilis of the liver. Death was probably due to sudden stasis in the region of the portal vein, which was particularly severely attacked. In two of the cases described there was ascites. (For peripylephlebitis, see p. 274.)

Spleen.

Enlargement of spleen is one of the most constant, and diagnostically the most important symptoms of congenital syphilis. The frequent enlargement of the spleen at a later period of infancy is considered to have a rachitic basis, which can hardly be held so in the newly born, so that enlargement of spleen in the first few weeks of life is regarded as a very characteristic symptom of syphilis when no septic disease is present. The spleen of syphilitic children is generally somewhat hard and protrudes very little beyond the costal arch. Much increase in the size of the spleen is certainly extremely uncommon in the newborn. Anatomically, it is a case of ordinary hyperplasia of the pulp. According to Schröder, myeloid areas are also frequently found in the splenic pulp. True splenitis is also supposed to occur.

Kidneys.

In foetal syphilis the kidney is very frequently sympathetically involved. According to Hecker, it is the most frequently attacked organ, as far as microscopic changes are concerned (60 per cent.). As in the foetus, the gross anatomical changes in the child that is born alive are, on the whole, of subordinate importance and are far less frequent than the microscopical changes (Hahn). Besides the circumscribed miliary gummata, the areas of softening (which we find in most internal organs) and "diffuse syphiloma" (as Steffen describes the diffuse round cell infiltration of the renal tissue extending from circumscribed cortical areas), it is principally the inflammatory manifestations which arouse our interest from the clinical point of view.

According to Hahn, in the syphilitic infant that is born alive, the following kidney diseases may be differentiated: the acute, sub-acute and chronic interstitial, and the acute parenchymatous and haemorrhagic nephritis.

On microscopical examination of the kidneys of syphilitic infants interstitial nephritis is relatively frequently found (Gallus, Karsvonen, Cassel). A more or less pronounced round cell infil-

tration of the interstitial tissue occurs, corresponding particularly to the connective tissue coats of the vessels, possibly with subsequent changes of the epithelia. Interstitial nephritis runs its course, as a rule, without any urinal changes or other symptoms indicating kidney disease. During life diagnosis is generally not possible. From the frequency of the pathological findings in the syphilitic infant and from the relative rarity of the same in an older child, one may conclude that the prognosis is favourable under suitable treatment at least in mild cases. In severe forms there is danger of a contracted kidney.

Acute parenchymatous nephritis is not frequently a symptom of syphilis. Finkelstein and Hahn have described three cases of haemorrhagic nephritis in children aged 7, 9 and 10 days respectively. The disease manifested itself by the appearance of oedema, albuminuria and haematuria. The children were somnolent, otherwise there were no uræmic symptoms. On palpation the kidneys were considerably enlarged. One case recovered, the other two died after a few days. The post-mortem showed the existence of haemorrhagic and interstitial nephritis. None of the children had received mercury before the outbreak of the disease; therefore mercurial poisoning was out of the question. Hahn associates the early appearance of the disease with the general experience, that the most severe internal organic changes occur at the end of foetal life and in very earliest infancy, whereas in the older infant they always become less frequent and milder.

In estimating the importance of milder grades of albuminuria with a view to the diagnosis of syphilitic kidney disease, one should be very cautious in the first week not to overlook the albuminuria of the newly born. On the other hand, it must be taken into consideration that parenchymatous nephritis is far more rare than the interstitial, and that the latter generally pursues its course without any epithelial changes, and accordingly also without albuminuria.

The author observed intense haematuria starting on the seventh day in a child with severe visceral syphilis. The post-mortem showed parenchymatous haemorrhages in the kidneys.

Suprarenal Capsules.

The suprarenal capsules are a frequent resort for the spirochaete, and in some cases they also show pronounced anatomical changes. There is either a typical proliferative inflammation, diffuse interstitial proliferation, or localized necrosis in the cortical substance (Guleke, Kokudo). Guleke attributes the existence of gummata not to changes in the vessels, but to intoxication of the parenchyma by the syphilitic virus. Esser had the opportunity of observing clinically two cases of congenital syphilis, in which (as proved by the post-mortem) the suprarenal capsules were chiefly attacked. From birth onwards the children suffered from periodical or constant vomiting, independently of taking food, also diarrhoea of a colicky

type. Esser attributes these manifestations to suprarenal insufficiency, as he observed similar symptoms in an older infant, in which the post-mortem showed residues of suprarenal hemorrhage.

Intestine and Peritoneum

Syphilitic diseases of the intestine are rare. Minck differentiates two forms of syphilitic enteritis. The changes may occur in the region of Peyer's patches (*syphilis annularis intestinalis*), from which diffuse infiltration of the mucous membrane may result, or there are irregularly scattered nodules, or small necrotic areas. Syphilitic enteritis originates obviously from disease of the vessels. Occasionally ulceration occurs. E. Fraenkel reports a case of chronic fetal peritonitis and congenital ulcerative syphilis of the small intestine, the latter leading to purulent peritonitis, from which the child died after five days; there were masses of spirochetes in the circular ulcers situated in the jejunum; otherwise the post-mortem showed no syphilitic organic changes, apart from osteochondritis, which was found microscopically.

Baumgarten also reports on a child with syphilitic intestinal ulcers and fibrinous deposits on the intestinal coils and liver; the child died immediately after birth.

The clinical symptoms of syphilitic enteritis are, as a rule, not very characteristic. In ulcerative processes there is, of course, a possibility of the discharge of purulent stools containing blood.

The disease of the vessels in the intestinal mucous membrane may give rise to severe hemorrhages, manifested clinically by melena. In advanced disease of the intestinal wall there is also the possibility of a spontaneous intestinal perforation.

The peritoneum is occasionally attacked by fetal syphilis (Simpson). This chronic fetal peritonitis may lead to formation of adhesions and ascites. The ascitic fluid sometimes contains abundant spirochetes (Gräfenberg).

Pancreas.

Interstitial pancreatitis is not infrequent in congenital syphilis. Owing to the premature development of abundant connective tissue in intra-uterine life it appears to lead to arrest of development or delay of maturity of the pancreas, which is expressed by complete lack of secretory glandular parenchyma. The islands of Langerhans are in such cases present in great numbers, and are closely connected with the small glandular canals (C. Sternberg). We have no observations concerning the clinical symptoms of such diseases in the period of infancy.

Nervous System.

In congenital syphilis the brain and meninges appear to be involved in the process of disease to a greater extent and more frequently than might be imagined from the clinical symptoms.

Meningo-encephalitic processes are often found, as shown by the researches of Ranke, Weyl, Tobler, in infants with hereditary syphilis. Among the cases examined by these writers were premature and stillborn children, and also those that died shortly after birth. The changes in the central nervous system occur in the form of a proliferative inflammatory process with a lymphocytic or plasmacellular exudate as a disease of the pia, which spreads irregularly over the brain (with thickening of the tissue), occasionally with small encephalitic centres in the cortical and medullary substance. Sometimes there are small subpial hæmorrhages. Schmeisser found in a syphilitic newborn child two small abscesses, involving pia and pallium, enclosed by a pyogenic membrane and containing abundant spirochetes.

The considerable anatomical changes do not appear, as a rule, to cause any pronounced nervous manifestations. Even in carefully observed cases the cerebral changes are generally associated with an accidental post-mortem finding. Weyl only saw a single instance (in an older infant) of a temporary eclamptic attack. Heine points out that syphilitic children sometimes show a marked restlessness and scream a great deal at night. He considers the screaming as the expression of meningeal irritation, and advises that in such children the ophthalmoscope should be used (see below). Lumbar puncture has been tried in order to establish the diagnosis, and in syphilitic children—the youngest was 7 days old—a marked and extreme degree of lymphocytosis has been found in a large majority of cases (Tobler). As the result of his researches, Baron came to the conclusion that a positive cytological finding was not a proof of syphilis. Lymphocytosis is not a proof of the existence of meningitis (Merzbacher); this is indicated by the circumstance that small lymphocytes are found in the spinal fluid, but large, round and plasma cells occur in the pial exudate. The amount of the albumen content gives no definite information. In a 4 months' child with clinical symptoms of hydrocephalus, Rach was able to establish the diagnosis of syphilitic meningitis by the finding of spirochetes in the opaque cerebrospinal fluid during life, but points out that the presence of numerous spirochetes in the inflammatory thickened meninges does not appear to be the rule. According to Hochsinger, the lumbar puncture fluid of syphilitic children that contains syphilitic anti-bodies, and shows a positive Wassermann reaction, is generally free from spirochetes. But in suspicious cases, spirochetes must always be sought for. Schridde found in the cerebrospinal fluid of a child of three days two spirochetes, besides cocci and bacilli. Spirochetes have repeatedly been found in the brain and spinal cord (Simmonds, Ranke, Hedrén, Dürck, Schmeisser).

Descriptions have also been given of gummatous meningitis of the brain and miliary gummata on the ependyma of the lateral ventricles.

The frequency of meningo-encephalitic changes in syphilitic children raises the question whether, in spite of the absence of

clinical symptoms at the time, sequelae may not occasionally occur in the future (mental abnormalities, &c.). But for this we have no definite evidence. As a rule the prognosis seems to be relatively favourable.

Owing to endarteritis of the cerebral vessels various disturbances of circulation and their sequelae may be found, but these generally become manifest at a late period of childhood. Ruch reports on a case of encephalomalacia of the frontal lobe in a child of three weeks, which began to snuffle in its second week and suffered from a sanguineous discharge from the nose and mouth, and finally could not take its mother's breast. The post-mortem showed softening of the whole left frontal lobe and thickening of the meninges, which contained a few spirilla.

Meningitis serosa interna and externa, which runs its course in the form of hydrocephalus, and, according to Hoesbinger, is the most frequent affection of the brain, is very seldom congenital. By the time the disease has developed the child has generally passed the first weeks of its life. This applies both to the slow developing and acute forms. But it is not impossible that many of the mysterious forms of convulsions in the newly born owe their existence to acute extravasations of syphilitic origin. Convulsive attacks may possibly be caused by pachymeningitis hæmorrhagica, such as not infrequently occurs in older infants with hereditary syphilis.

As in the cerebral meninges, diffuse interstitial inflammation of the connective tissue and cell proliferation may be found in the membranes of the spinal cord on the one hand, and on the other military gummata. Sibelius describes disturbances of development of the spinal ganglia in syphilitic newly born, conditions which Zappert regards as manifestation of hereditary syphilis, though not unreservedly. In a syphilitic stillborn child Fourner found diffuse infiltration of the spinal cord with embryonal tissue. According to Gasne and Gilles de la Tourette, syphilitic myelitis is found in the newborn and also in the older child always secondarily, combined with cerebral syphilis or syphilitic periostitis of the vertebrae.

R. Peters describes paralyzes of spinal origin. They sometimes occur in the newly born, though more seldom than in the older infant. They are sometimes total, sometimes partial flaccid paralyzes of one or both extremities, which occasionally assume the peculiar fin-like appearance. The paralysis may also attack the cervical musculature and the lower extremities, and be combined with secondary contractures. Peters attributes the disease to syphilitic arterial changes with scattered foci of softening and formation of scar tissue, but with suitable treatment the prognosis is generally supposed to be favourable. Anatomical proof of such syphilitic changes in the spinal cord has hitherto not been furnished.

Schreiber associates those cases of Parrot's paralysis, where no osteochondritis is found, with toxic influence of either the syphilitic

toxin or the toxin of the septic organisms in the blood (mitigated streptococci) on the ganglion cells of the spinal cord and the spinal ganglia.

Eyes.

The anatomical and histological examinations of the eyes of syphilitic newborn children (Rab, Schlimpert) have proved that, in accordance with the blood-supply of the eye, the choroid contains abundant spirochaetes. From here the latter pass to the iris, the cornea, and to the lower sclerotic layers. In abortive fetuses Reiss found parenchymatous keratitis and iritis (see p. 358). Spirochaetes and inflammatory changes are sometimes found in the conjunctivae (conjunctivitis gummosa), in the lachrymal glands and eye muscles. Squinting, so repeatedly observed in syphilitic infants, may possibly be associated with specific myositis.

The optic papilla seems to be relatively frequently affected. The disease probably extends from the cerebral meninges. It may perhaps be assumed that the frequent occurrence of changes in the optic nerve and retina, which ontogenetically represent part of the brain, is associated with the frequency of the above mentioned syphilitic meningo-encephalitic changes (Japha). Optic neuritis, for which there is hardly any other cause in young infants than syphilis, is found, according to Japha, in about 66 per cent. of cases of congenital syphilis, and according to Heine, in about nearly 83 per cent. Heine points out that the examination of the fundus oculi in syphilitic infants that scream a great deal nearly always means optic neuritis. The latter sometimes precedes the appearance of other syphilitic signs by some weeks. If other symptoms of syphilis are absent ophthalmoscopic examination is of great importance diagnostically. The youngest child, in which optic neuritis was found, was 13 days old.

Hirschberg describes the changes of the fundus oculi as follows: Ill-defined, and cloudy optic disc and surrounding retina. Bright spots in the fundus oculi, which increase in number and size in course of time, and finally show pigmentation. The centre of the retina soon shows a grey colour, but which later on may fade somewhat. In the periphery are found dappled or dark centres. The disease is always bilateral, but not invariably equally severe on both sides.

Ears.

Clinical evidence of syphilitic ear diseases does not occur in the newly born. Otitis media is not a frequent complication of syphilitic rhinitis and hardly ever of specific origin. According to Mayer's researches, specific processes in the meninges may be combined with neuritis of the eighth nerve even in children during the first days of life. The process is further transmitted through the lymph passages to the internal ear.

Skeleton.

One of the most important anatomical signs of syphilis is syphilitic osteochondritis. It often renders the anatomical diagnosis possible if other symptoms pointing to syphilis are absent, whether during life in the integument, or post partum in the internal organs. It is often found in those forms described as latent syphilis.

Syphilitic osteochondritis is situated at the junction of the epiphyses and diaphyses of the long, medullated bones, and also at the osteochondral junction of the ribs. It leads to widening and irregular formation of the zone of calcification which is indistinctly outlined against the epiphysis, and sends out jagged offshoots into the latter. In advanced cases a mass of granulation is produced in the form of a broad yellow layer containing connective tissue and necrotic foci which sends out tongue-shaped processes towards the epiphysis, and lends a jagged appearance to the otherwise straight yellow boundary zone. More or less abundant spirochaetes are found in the affected parts. In severe cases a condition of diminished resistance is created, which, in otherwise insignificant traumas, may give rise to separation of an epiphysis. This may happen before or during birth. Muscular contraction also plays an important part.

If the surrounding periosteum is not attacked and no separation of the epiphysis is present, the clinical diagnosis of osteochondritis is only made possible by X-rays, by means of which the broad and jagged condition of the zone of calcification may be made clearly visible. Osteochondritis is very frequently combined with infiltration of the periosteum and surrounding soft parts, and leads to spindle-shaped distension of the parts attacked. These distensions are frequently found in the region of the inferior epiphysis of the humerus. As a rule they are very painful and are connected with a very striking clinical group of symptoms, the so-called Parrot's pseudo-paralysis.

Occasionally this paralysis—and this is diagnostically important—may appear as the first perceptible external symptom of syphilis, and in other cases it appears simultaneously with the appearance of a syphilitic eruption. It may be recognizable shortly after birth, even on the first day. According to Scherer's statistics, this was twice observed in fifty cases. Such cases may be mistaken for obstetrical paralysis; usually it develops later, according to Hochsinger, between the first and fifth week. The pseudo-paralysis may start gradually or suddenly. In the typical picture of a Parrot's paralysis the affected arm is not held in the elevated position favoured by small children, but lies loosely by the side, generally adducted, and the forearm somewhat pronated. If the arm is raised and let fall, it drops heavily back on to the pillow. Passive movements, also active movements, which can generally be produced by needle pricks, &c., are accompanied by loud cries of pain. The painfulness may also be observed before the characteristic position has developed, and is therefore an important premonitory symptom, but it must be pointed out that in some cases pain is absent or very slight.

Swelling generally develops later. If separation of the epiphysis has occurred it may be established sometimes by crepitation. The epitrochlear lymph glands are frequently enlarged.

According to Hochsinger's theory this paralysis is the result of an affection of the muscles, which in most cases is probably due to the inflamed state of the periosteum (polymyositis syphilitica). The Röntgen rays show that in some cases the infiltration chiefly involves the musculature. This is associated with interstitial or parenchymatous degenerative changes in the muscles. The nerves are always intact. The term pseudo-paralysis is therefore quite justifiable.

With correct treatment the prognosis of Parrot's paralysis is quite favourable.

Diffuse periostitis of the long medullated bones and syphilitic changes of the vertebral column, which are found in the early months of foetal life, can hardly be established clinically. Examination by Röntgen rays is the only possible means of diagnosis.

Affections of the joints are only exceptionally observed. Suppurative arthritis, which occasionally occurs, is considered by Heubner and Hochsinger as a secondary infection. Whether this theory applies to all cases is doubtful. Marfan observed an infant with purulent exudate in both knee joints; the pus was free from microbes; with mercurial treatment the condition completely healed.

Parasyphilitic Manifestations.

Foetal syphilis of the internal organs (kidneys, liver, lungs, pancreas, &c.) is not only associated with inflammatory processes, but also leads to disturbances in their development and tissue formation. Like tuberculosis, syphilis may harm the child, probably by the influence of its toxins, without actually infecting it. In this way disturbances of development sometimes occur, as also anomalies of constitution and symptoms of deficient vitality, without specific organic changes. Syphilis in the parents is frequently held responsible for malformations.

The Clinically Latent Form of Hereditary Syphilis and Acquired Syphilis during the Newborn Period.

As already mentioned, the clinical symptoms of syphilis frequently appear after several weeks of apparent health. The cause of this latency may be of various kinds. Of course, in many cases there may already be syphilitic changes in the internal organs which pass unnoticed; the early stages of osteochondritis, in particular, occasionally at the post-mortem may be found to be the sole but certain sign of an already existing syphilitic disease. In other cases the child suffers from birth from rhinitis or shows a slight enlargement of the spleen. But in many it may be assumed that the infection has occurred during the last few days of pregnancy or

during birth, and that there are actually no syphilitic organic changes present in the newborn child.

Grafenberg believes that, especially if no spirochetes are found in the umbilical cord, these are cases of syphilis acquired during or after parturition, and not an infection transmitted through the blood. He is of opinion that particularly the nasal mucous membrane may be the seat of a primary affection. In support of his theory he publishes the following case: Mother with condylomata on the vulva; umbilical cord free from spirochetes; child healthy. Wassermann negative. After three weeks syzyza, Wassermann positive. Haslund reports a very rare occurrence: A child, whose parents were both syphilitic, acquired syphilis only at birth, and a primary sore developed in the form of a wound on the face, made by the forceps at birth.

However syphilis may be transmitted in such cases the question arises whether it is possible to prove that the newly born child is infected or not. The question appears to be important, since it happens occasionally that a child of syphilitic parents remains permanently healthy, particularly when the transmission of syphilis from the mother to the hitherto healthy woman occurs at a late stage of pregnancy (post-conceptual syphilis). It is well known that the thesis has been advanced that the apparently healthy children of syphilitic parents are immune to syphilis (Proden's law), but belief in the general application of this law has somewhat declined. One circumstance alone contradicts the theory of transmission of an immunity from the affected mother to the child, viz., that in the final stage of pregnancy and during birth transmission of the disease to the fetus is possible, therefore at a time at which the transmission of immunizing substances must naturally have taken place. If these healthy children of syphilitic mothers are not affected, the reason probably is that the possibility of infection, even if the child is nursed by the mother, is extremely slight, as in cases where syphilitic changes are present on the nipples.

Is it, therefore, possible to recognize an infected but clinically healthy child in the incubation stage? As a rule the question must be answered in the negative. The Wassermann reaction in such cases is generally negative (Halberstaedter, E. Müller and Reiche, Rietschel). Thomsen and Boas have made the important discovery that the placenta and umbilical cord of these children, who after birth give a negative Wassermann reaction but are later proved to be syphilitic, may show syphilitic changes. The Wassermann test, therefore, in the newborn child should be combined with an examination of the umbilical cord and placenta. The condition of the Wassermann reaction in the mother may also give a certain clue. Children of syphilitic mothers that show no signs of syphilis immediately after birth remain far more frequently healthy if the blood of the mother shows no positive reaction. (This only applies if the mother has not just previously had anti-syphilitic treatment.) There are newborn children who after a few weeks

show symptoms of syphilis, and in whom the Wassermann reaction proves negative; on the other hand, there are children who react positively at birth without showing any syphilitic manifestations later. In such cases the reaction disappears shortly after birth. Presumably one is dealing with a case wherein transmission of reacting substances has occurred from the mother to the fetus (Thomsen and Boas, Pillon). A negative reaction of the umbilical blood or of the child's blood does not prove that the child is actually free from syphilis and will remain so; but again a positive reaction is not always proof of infantile syphilis. In any case it is gathered from researches up to date that the Wassermann reaction, which proves constantly positive with the manifestation of symptoms, or shortly before this period, does not give any assistance in the clinically healthy newborn child for the diagnosis of latent syphilis, that is to say, for the recognition of an infection which has already taken place.

The Question of Suckling.

Colles-Baumes' law states that the healthy mother of an hereditary syphilitic child, who is permanently free from symptoms of syphilis, is immune to it. Protela's law states that the healthy child of a syphilitic mother is immune to syphilis. But we know that, on the one hand, this latter law does not entirely hold good, and that, on the other hand, many exceptions to Colles' law have been published. It is therefore not clear whether suckling is permissible, if only the child is diseased, but not the mother, or vice versa. The question of suckling is of the utmost importance. For in the prognosis of hereditary syphilis the feeding plays an enormous part, apart from anti-syphilitic treatment. One may justly and rightly maintain that the introduction of artificial feeding may aggravate the prognosis to a great extent even with the best treatment.

If both mother and child show signs of syphilis suckling should, of course, be allowed. If the mother is free from symptoms of syphilis, but not the child—a case which relatively frequently happens—then undoubtedly in the majority of cases the mother, according to Colles' law, is safe from infection. But if the Wassermann reaction is positive in the mother, suckling should, of course, be allowed. If it is negative, there is always the possibility that it may be one of the rare exceptions to Colles' law. According to Bab, if the mother's blood contains neither antigen (latent syphilis) nor antibody (immunity) suckling should be forbidden. But it is another question whether this should actually be done. In such cases it is generally not a question of severe syphilis in the child. If syphilitic lesions on the child's lips are absent, the danger to the mother in suckling is hardly worth mentioning. But if there is any reluctance, or the child is suffering from a highly infectious form of syphilis, the milk may be pumped off and given by the bottle, a procedure which, by half emptying the breast,

ensures sufficient nourishment at the beginning, or at least forms the basis of a later mixed feed. The application of a nipple shield is worth a trial in such cases.

If the mother is syphilitic, but the child free from syphilis, it may be latently syphilitic or healthy. If the latter is the case, then, in consideration for the probably numerous exceptions to Profeta's law—the birth of a healthy child with florid syphilis of the mother, a truly rare event—there is the possibility of infection from the affected mother. As the Wassermann reaction may be negative, not only in healthy children, but also in those who show symptoms of syphilis a few weeks later, general prohibition of suckling would rob the children, in such cases, unnecessarily of the natural food so important for their welfare. Suckling cannot be forbidden on the ground that it may be a case of the one child that remains permanently healthy. If actual syphilitic lesions are not present on the nipples, the danger to the healthy child should also be slight. The milk itself is always harmless. Spirochetes have never yet been found in it. According to Bal, it contains abundant antibodies, specific protective substances, which may be considered as actual medicine for the infant.

It may be pointed out here, that the administration of pumped off mothers' milk, which in living-in institutions is taken from mothers clinically free from syphilis, presents hardly any danger of infection to the children, even though one or other of the donors is latently syphilitic. In any case it may be safely asserted that the milk of a woman who is negative to the Wassermann test is fit for feeding healthy children, under all circumstances.

One may safely say that neither syphilis of the child for the mother free of symptoms, nor syphilis of the mother for the child free of symptoms, forms any contra-indication to suckling, and that, at the most in isolated cases, temporary feeding with pumped-off milk or the application of nipple shields, is indicated instead of direct application to the breast.

Prognosis and Treatment.

With natural feeding and suitable treatment there is a favorable prognosis for an older infant with syphilis. With syphilis neonatorum, which is so frequently combined with severe visceral changes and which so often attacks premature debilitated children, the prognosis is far more grave. Especially children with severe pemphigus die within the first few days or weeks, even with immediate antisyphilitic treatment and feeding with mothers' milk. Cases of maculo-papular or papulo-bubulous syphilis offer a much better prognosis, provided that the internal organs are not too severely affected.

As soon as the diagnosis of syphilis is established, antisyphilitic treatment should immediately be started. The question, whether healthy children, or those free of syphilitic symptoms, of syphilitic

parents should be treated prophylactically, is answered by Hochsinger in the negative. He only recognizes an indication for the antisyphilitic treatment in manifest syphilis. Even when the child is clinically healthy Baisch commences antisyphilitic treatment as soon as the child's blood gives a positive Wassermann reaction.

Mercurial treatment.—Two methods only come under consideration, viz., internal treatment and injection. The favourite remedy at the present time is hydrarg. iodi. flav., which is given three times daily to the newly born, in doses of crongron . The other mercurial preparations (calomel, hydrarg. tannicum oxydul.) are superfluous and apparently inferior to the protiodide. The injection method is best executed by a 1 per cent. sublimate solution, 0.1 c.c.m. (= 0.001 gm. sublimate), the contents of a marked division of a Pravaz's syringe being injected intra-muscularly. The injections are made once or twice a week. In children who swallow badly, this method, allowing of an exact dosage, has much to recommend it. With weeping forms, especially those exanthemata associated with vesicular eruptions, sublimate baths may be administered with good results (1 gm. = 1 sublimate pastille to a bath of about 20 litres). If the eruption is very widespread and the absorbing surface is relatively large, caution is then necessary owing to the danger of sublimate poisoning (intestinal hemorrhage). Inunctions are not applicable to the infant, particularly the newly born, owing to the delicacy of the skin. Even the application of small pieces of grey plaster on the breast or back must be carefully done, as folliculitis may easily occur.

In rhinitis, treatment with ointment (1-2 per cent. yellow or 5 per cent. red precipitate ointment) is of value. The ointment is introduced into the cleansed nostril by means of a cotton-wool plug, provided with a thread, and left there for one hour (Hochsinger). For severe swelling and obstruction to breathing the administration of adrenalin is recommended (instillation of a solution 1 : 5,000, or an ointment). Epiphyseal swellings at the extremities may be covered with a narrow strip of grey plaster.

Salvarsan treatment.—In certain cases of the newly born, this treatment seems to be vastly superior to mercurial treatment. Baisch points out, that even in malignant forms of congenital pemphigus, which hitherto have defied all treatment and have almost invariably a fatal termination, quick recovery may occur. The immediate success of salvarsan injection, is, according to the author's experience, extremely striking. Even on the following day, the further eruption of the exanthem may suddenly cease, the vesicles dry up, the papules decrease in size and quickly fade away. The occurrence of relapses, however, can hardly be prevented. If, after a while, one does not wish to repeat the salvarsan injection, it is advisable to introduce prophylactically a mercurial treatment. This combined procedure has been most satisfactory, even with the young infant.

One must not be too timid with the dose of salvarsan in the newly born. The dose of 0.005 gm. per kilogram of body-weight,

which was at first recommended, may well be exceeded. Baisch injects as much as 0.15 gm. Engelmann recommends amounts of 0.04-0.1 gm. and lays stress on the fact that in severe infections not less than 0.04 should be given. As a minimal dose for a newborn child the amount should be fixed at about a half a decigram.

As to the method of administration, the intramuscular and intravenous may be considered. The latter procedure is, of course, the ideal one. Engelmann suggests the following method: Constriction of the arm by means of a thin rubber tube, exposure of a cubital vein, insertion of a strong cannula which is connected with a 20 c.cm. Luer's syringe by a thin rubber tube. The cannula is firmly held by one person's hand, while another presses the liquid slowly into the tube by turning movements. The preparation is administered in physiological salt solution (0.04 : 20). With well-developed veins in the skin of the scalp, a vein of the head may also be used for injection. The intramuscular (intragluteal) injection has the disadvantage that it frequently forms a dense infiltration or a necrosis results and that with the discharge of the broken-down infiltrate externally there is danger of a secondary infection. But if intravenous injection is not possible, for technical reasons, these advantages need not further be considered. As a rule the infiltrates are absorbed and with suitable dressing secondary infection may always be avoided. For intramuscular injection emulsion of salvarsan in sterile oil is best used, this being prepared with a sterilized pestle and mortar. The quantity of oil used should be as little as possible (5-10 c.cm.).¹ In view of the thickness of the oily emulsion a cannula with a wide lumen should be used (such as a lumbar puncture needle) and an absolutely efficient syringe (Record syringe).

¹ Postscript.—A considerable simplification of the method is afforded by the use of concentrated salvarsan solution, in particular neo-salvarsan, soluble in water. Welde (*Verhandl. d. Ges. f. Kinderheilk.*, 20, Münster, 1912) uses for infants solutions of 0.5 gm. in 2 c.cm. injection fluid. For salvarsan the latter consists of 1 c.cm. freshly distilled water + 1 c.cm. physiol. NaCl solution + 0.5-0.8 norm. NaOH; for neo-salvarsan, of 2 c.cm. fresh Ag dist., so that the whole solution may be contained in a small syringe containing 3 c.cm. Welde recommends injection into the cranial veins as the best method of administration. By using concentrated neo-salvarsan solution prompt absorption may be expected after intramuscular injection.

CHAPTER III.

SEPSIS.

Meaning of the term. Portals of Infection.

The term "sepsis" is somewhat wide and indefinite. It is usually applied to those bacterial general diseases, the excitants of which primarily belong to the group of common pyrogenic organisms, but which may be caused by various other micro-organisms which enter the body by some one or other entrance and are

diffused in the organism by way of the blood or settle in the organ corresponding to the inlet of the infection. From thence toxic products of their metabolism enter the blood. Septic diseases therefore are divided into two groups, general infections caused by bacteraemia and those caused by toxæmia. And again in the first group may be differentiated those diseases in which the bacteria, in the blood, merely produce a general toxic effect, and those in which the excitants settle in the various organs in circumscribed places and there form multiple metastatic centres of disease. In the latter case one is dealing with suppurative foci, and pyæmia is the term used.

From a theoretical point of view the term sepsis should include all those diseases caused by microbes, which are combined with general symptoms. But if this is not done and a large number of diseases is excluded, it is because one is guided by a clinical point of view. Those diseases which are distinguished by a characteristic, clinical group of symptoms, diseases to which in many cases a definite "specific" excitant corresponds (e.g., typhoid, influenza, &c.) are not generally counted as sepsis. Accordingly the so-called specific causes of disease are not reckoned among the real causes of sepsis. Septic micro-organisms cause no specific types of disease, but are very varied in their clinical picture, such as is particularly the case with streptococci and staphylococci, and with these is associated the pneumococcus. Furthermore, the general infections caused by *Bacterium coli*, the *Bacillus pyocyaneus* and Friedländer's bacillus (*Citrus*) are also counted as sepsis. The term is thus made much more comprehensive in an aetiological respect. The group of *Bacterium coli*—one generally speaks of the coli-typhoid group—is very extensive and includes many kinds and varieties, paracoli and paratyphoid bacilli, &c.

From the abundance of aetiological possibilities and the variety of places of entry for infection, it follows that that which we term sepsis is a very wide collective term, which, strictly speaking, means merely a general disease on a bacterial basis. But if this is nevertheless retained it is because one is not in a position to distinguish the various aetiological factors according to the clinical forms and, for instance, to distinguish clinically streptococæmia from staphylococæmia or colibacillæmia and the corresponding toxæmic. The poisons affect, though to varying extent, the same organs in the same injurious manner, and this results in a certain similarity of the clinical picture, at least as far as the general symptoms are concerned.

The infection of the child by septic organisms may occur during intra-uterine life, intra partum or post partum.

Intra-uterine infection occurs either through the placenta or liquor amnii. The injuries which the fœtus suffers, in septic diseases of the mother, would probably be due, less to transmission of bacteria from the maternal blood to the foetal circulation, than to injury from their toxic substances. On the one hand bacteria by no means always circulate in the blood of the affected mother,

and on the other hand the placenta forms a barrier. It was repeatedly asserted while discussing the intra-uterine transmission of acute and chronic infectious diseases, that this barrier may be pierced. The conditions with septic organisms are quite similar.

Concerning the frequency of death of the foetus, in infectious diseases of the mother, the foetus generally dies from the consequences of intoxication or from insufficient nutrition owing to the maternal disease. According to Schmuddecker a part of the toxin is transmitted from the maternal blood to that of the foetus and causes the same disturbances of function in the child's as in the mother's organs. In these cases we should be dealing with a toxæmia, transmitted *in utero*, without transmission of bacteria. But also in those cases where pathogenic micro-organisms are transmitted through the placenta from the mother to the foetus, the infection of the foetus does not lead straightway to a specific disease. During the early embryonic stages of development the foetus appears to be protected by an immunity against the true disease of the mother—an immunity which may possibly be attributed to a deficiency in specific cell receptors. This immunity disappears slowly towards the end of foetal life. Only in the last two months of pregnancy do pronounced diseases of the foetus occur (B. Fischer). But that also at this time the disease may progress comparatively mildly, follows from the fact, that focal inflammatory conditions which are due to intra-uterine transmission of disease germs, however severe they may be in their consequences for the child, are at the time of birth generally in the process of recovery or else completely healed.

Septic diseases of the mother, therefore, may injure the foetus in many ways, either through harm to its development from deficient nutrition, through transmission of toxins from the maternal to the infantile circulation or through infection with the organisms of the disease. Strictly speaking, only in the latter case is one justified in speaking of sepsis.

Sepsis may be transmitted in the uterus through the placenta or liquor amnii. The significance to be attached to the placental mode of infection is hard to decide.

As Runge says, in spite of all our up-to-date methods which might give an answer to the question, the material is very scanty, since, thanks to modern aseptic methods, septic diseases during pregnancy and birth are very rare occurrences. According to information dating from pre-aseptic times, in a period in which "puerperal fever of newly born" was a frequent occurrence, suppurative pleuropneumonia was a frequent condition in those children who succumbed to placental infection (Weber). Buhl found in stillborn fetuses a gelatinous serous infiltration in the connective tissue round the umbilical vessels which extended through the subperitoneal tissue to the vertebral column, the connective tissue of the mediastinum and the inter-lobular tissue of the lungs. Orth mentions a case of pulmonary abscess and empyema, which he was

able to prove to be caused by placental infection in a child of a septic mother, that died between its third and fourth day. Runge, who critically sums up many earlier observations, is somewhat sceptical as to their indicating a placental mode of infection.

Those conditions arising through infection of the liquor amni are much more distinct. The liquor may become infected, not only after the rupture of the foetal membranes, but bacteria may penetrate through the intact foetal membranes; from the peritoneum the bacteria may pass through the ribs, reach the foetal membranes, infiltrate the latter and thus reach the liquor amni (Hellendall).

The danger to the child in maternal pertyphilitis is probably thus explained. Hellendall points out that in those cases (about 110) described in literature, of pertyphilitis during pregnancy, the children were either born dead or died shortly after birth from septic disease.

The danger to the child from infected liquor amni may be gathered from the following statistics of Lehmann. In 50 births with foul-smelling liquor amni, 29 were born dead (or so asphyxiated that they could not be resuscitated), 4 died during the first few weeks and only 20 survived. According to other statistics the mortality of the children is 18, 19, 22, 43 per cent.

In these cases the principal entrance for infective organisms appears to be the lungs. This is shown by the relatively frequent occurrence of pneumonic areas in post-partum mottified fetuses. In contrast with infections due to aspiration, those arising from infection of the umbilical cord and digestive tract are of minor significance (Hellendall). But Runge is of opinion that these cases of pneumonia may frequently be regarded not as a primary focus but as a metastasis from sepsis, usually of umbilical origin.

During birth infection may ensue from transmission of maternal vaginal germs to the child. In these cases there is generally at first a local condition, which, like sepsis acquired post partum, may form a point of origin for a general affection. Bacteria of the vaginal secretion may get into the child's mouth and from thence, either directly or through stomatitis, they may penetrate the body; they may be swallowed or aspirated and infect the lungs, or be absorbed into the organism through the intestinal canal or through injury to the skin. At the moment of birth the basis for umbilical sepsis may also be laid. The finding of similar organisms in the maternal vagina and in the child tends to prove the actual occurrence of this mode of infection (Liedemann and Noack).

However significant may be those infections in utero and the so-called "obstetrical" infections with the birth of the child through the infected genital passage, they are of minor significance compared with extra-uterine infections. The latter may derive their origin from various parts of the body. At the place of entrance the bacteria may cause abnormal changes, or penetrate the body without any clinically obvious local affection, therefore without any recognizable primary affection (cryptogenetic sepsis). The most popular

entrance, if one may so call it, for septic affection of the newborn is formed by the navel. In the chapter on affections of the navel and umbilical vessels the manner in which general infection may result has already been discussed. It may be repeated again here particularly that umbilical phlebitis nearly always leads to immediate general sepsis, whereas in local affections of the navel and in arteritis, a certain period as a rule elapses before the general infection appears; and, furthermore, it may once again be emphasized that umbilical sepsis may be present without obvious external changes at the navel, or at least such as would correspond to the severity of the general affection.

That an infection may result through the physiological wound of the navel may easily be conceived. Many medical men have therefore adopted the erroneous theory that sepsis in the newly born is practically always umbilical sepsis. This must be definitely contradicted. The wrong idea of the predominating importance of the navel as an entrance of infection in infantile sepsis easily gives rise to an extremely one-sided prophylaxis, which is most insufficient for the child's welfare. It is immensely important that medical men and midwives should learn that the excitants of disease may penetrate the infant's body by many other entrances.

The infection may occur from various parts of the external surface of the body; from injuries to the skin, which occur during birth; from erosions and superficial loss of epithelium; from decubital abrasions, such as are sometimes found with restless children, above the internal malleoli. In this respect intertrigo plays a certain part which must not be underestimated. From all the above lesions, harmless in themselves, local processes of supuration (abscesses, phlegmon) may arise and general sepsis may follow. In the same manner all originally local, infective skin diseases (pemphigus) may form the basis for a general affection.

As regards the external mucous membranes principal significance must be attached to the oral mucous membrane as an entrance for sepsis. The septic oral affections have already been discussed; palatal spots, palatal ulcers, stomatitis and their resulting conditions form the places of origin, from which the septic microbes may infect the entire system. The bacteria may penetrate the salivary glands (without any visible change of the oral mucous membrane) and cause inflammatory processes. Finally the possibility must not be excluded of a general infection from the oral mucous membrane, which has been either injured or made permeable even without a primary lesion.

Next to the oral mucous membrane in importance are the nasal and pharyngeal mucous membranes. The general infection may proceed from a coryza or rhinitis posterior. The pharyngeal tonsil is of far greater significance in the newly born than the palatal tonsil. The middle ear may also be the seat of the primary affection. From the nose, throat or ear infection of the meninges may arise and meningitis thus resulting, may be the starting point of the

sepsis. The conjunctivæ and vulva very rarely form the entrance for general infections.

The mucous membranes of the respiratory and particularly of the digestive tract must be considered as extremely important, but are very much under-estimated as starting points of a septic affection. The importance of bronchial infection and pulmonary sepsis has already been pointed out by Fischl. He draws attention to the fact that infection from the air may play an important part in hospitals. The septic organisms may be derived from the lochial secretion of infected lying-in women; even in the dried condition they preserve their virulence for a certain period, and they become mingled with the dust and inhaled by the children. If they penetrate the lungs bronchitis with epithelial necrosis or pneumonic foci are produced and spread further along the lymph channels. In this manner Fischl observed streptococcic, staphylococcic and pneumococcic infections arise. From a diagnostic point of view it must be remarked that only the early appearance of clinical pulmonary symptoms at the beginning of a general affection points to the presence of infection caused by inhalation. The result of the post-mortem alone is not a criterion, as pulmonary affections may easily exist secondarily in a weakly resisting system or may arise metastatically.

Czerny and Keller in particular attribute much importance to enterogenous sepsis in the newly born. They support their theory by observations which have shown that the intestine of the newly born may be permeable by those bacteria, which the intestinal wall of older infants, children and adults or of experimental animals does not permit to pass. Just as in comparatively slight disturbances of the general condition thrush may settle in the oral cavity of the newborn child, a relatively slight decrease of resisting power of the whole system is sufficient for the intestinal mucous membrane to lead to tissue change which will render possible the entrance of bacteria. These statements are principally supported by the well-known results of Czerny and Moser who found micro-organisms in the circulating blood of children in the first weeks of life who showed clinical symptoms of a general infection. Czerny and Keller attach immense importance to these bacterial conditions. They are of opinion that the suppuration in fractures which may occasionally be noticed in septicly infected children after uncomplicated birth injuries, are caused by bacteria emanating from the interior of the intestine and circulating in the blood; they agree with Kader's theory on the nature of the hematoma of the sternocleidomastoid muscle and attribute the infection of the muscular wound to enteral infection; they are also inclined to regard many umbilical vessel affections as secondary localizations of a general infection, proceeding from the digestive tract in a *locus minoris resistantiæ*. Finally, when Czerny and Keller also associate icterus neonatorum, melæna and nephrosis with enteral sepsis, the enormous importance which they attach to enteral infection in the pathology of newborn is thus shown.

It is hardly generally acknowledged that such predominant importance should be attached to infections proceeding from the interior of the intestine, in the pathology of the first few days of life. But that the digestive tract, as an inlet for septic diseases, has a much greater significance than has hitherto been generally accepted, is a fact beyond any doubt. The injury which renders the intestinal mucous membrane permeable for bacteria may be caused partly by the invading microbes themselves (in which case the relation between the power of resistance of the organism and the virulence of the bacteria comes under consideration), and partly the alteration in the mucous membrane caused by the physiological irritative catarrh may also play some part. The diagnosis of the intestinal origin of a septic disease certainly meets in isolated cases with difficulties, and many a cryptogenetic sepsis may come under this category. Gastro-intestinal symptoms need not necessarily occur, their absence is as little against the existence of a septic general disease of intestinal origin as their presence may be considered pathognomonic of the latter.

Other substances of intestinal origin besides bacteria may be responsible for the appearance of severe general symptoms. The intestinal auto-intoxications of later life, which may probably be attributed to the absorption of toxic products of decomposition, which are due to the activity of pathogenic bacteria, may also find their analogies in the pathology of the newborn. Ganghofner and Langer have shown that in the very first few days of life the gastro-intestinal tract is permeable by true albuminous bodies. If this is actually the case, other high molecular compounds would also be able to penetrate the intestinal wall of the newly born. Accordingly, it should be expected that during the first few days of life, not only an enterogenous bacteriæmia, but also enterogenous toxæmia and auto-intoxication would comparatively easily occur.

Unfortunately very few well-founded clinical facts correspond to the abundance of possibilities. Chemical results which might give information regarding the nature of the toxic factors are almost entirely lacking in enterogenous infections and intoxications of the newly born; the bacteriological results are also mostly unconvincing. If during the course of a septic disease streptococci are found in the stool, this is always taken as a proof of its enteral origin. In staphylococcal conditions the pathological significance of bacteria found in the stool is much smaller. In those general infections classified as coli sepsis, in the absence of another entrance, the theory of enteral origin is the most likely; but the diagnosis can only then be confirmed by the presence of bacteria in the blood. Exact bacteriological examinations, not merely in the testing of smear preparations and the ordinary culture smears, but also studies of chemical processes in the interior of the intestine, are urgently required for the furtherance of our knowledge in respect of intestinal sepsis and intoxication.

The septic organisms comparatively seldom penetrate the body

of the newborn by way of the genital tract or through the urinary passages, which at a later period of infancy relatively frequently form portals for general infection.

The newborn child is very susceptible to septic infection. The bacteria find relatively easy admission into its body, through the physiological umbilical wound, the easily vulnerable mucous membranes, the delicate desquamating skin, the true horny layer of which is still absent, and through the functionally incomplete intestine, &c. The bacteria and toxins easily become disseminated in the body, as the phagocytic activity of the leucocytes is still deficient. Enlargement of the lymphatic glands which, to a certain extent, check the infection, is generally absent in the newly born or very slight. Its natural power of resistance against septic organisms is particularly small. A deficient formation of protective materials might also be responsible for the dissemination of the morbid agents.

Clinical Symptoms, Prognosis and Treatment.

The symptoms of septic infections may be described as local and general. The local symptoms proceed from anatomically circumscribed morbid changes in individual organs and correspond partly to the primary focus of disease, from which the sepsis either arises directly or after spreading in the vicinity, and partly to metastatic pyæmic affections which are evoked by the bacteria, circulating in the blood, in distant parts of the body. The symptomatology of these local diseases has been discussed in the description of the pathology of the individual organs and systems. We will therefore only dwell briefly on the septic organic diseases and refer for the rest to the chapters in question.

Digestive tract: Septic diseases of the oral cavity, stomatitis, inflammation of tooth pulp, purulent sialadenitis. Septic gastroenteritis and melæna, peritonitis.

Respiratory tract: Rhinitis anterior and posterior, otitis, bronchitis, pneumonia, pulmonary abscess, purulent pleurisy.

Circulatory apparatus: Purulent pericarditis.

Urogenital tract: Pyelocystitis and nephritis, nephrosis, vulvitis, septic menorrhagia.

Nervous system: Meningitis, encephalitis.

Skeleton: Osteomyelitis, purulent arthritis.

Skin: Abscesses, phlegmons, pemphigus and other infective exanthemata. Mastitis.

Navel: Inflammatory local affections (affections of umbilical vessels).

The local affections present their characteristic symptoms and give the special features to the individual case. That which we term the septic components of the clinical picture does not refer to the primary or metastatic local affections, but to the general symptoms which are caused by the flooding of the system with toxic

substances. Their effect is seen at the autopsy by the general diffuse degeneration of parenchymatous organs.

The most important though very indefinite signs of sepsis consist in the changes of the general condition and the external state of the body. The children assume a peculiar shrunken appearance. The facial expression is tired and apathetic. Instead of the healthy crying of children there is painful whimpering and groaning or complete stillness. The turgor of the skin is deficient and the colour anæmic and grey; if jaundice is present the skin shows a faded yellow colour, characteristic of sepsis. It is very difficult to decide in the individual case how far it is a case of icterus neonatorum influenced by the septic anæmia, and how far it is a case of genuine septic icterus from abnormal changes in the liver (see p. 61). Frequently a slight cyanotic discoloration of the distal parts of the body is also added, sometimes sclerema and sclerodema. Owing, on the one hand to toxic breaking down of tissue and loss of fluids, and on the other hand to insufficient nourishment and fluid, rapid emaciation and exsiccation will occur. The clinical picture which finally develops, without presenting any pronounced individual symptoms, possesses very characteristic features. It is known as "habitus septicus."

Septic fever which is so characteristic of sepsis in later life, is an inconstant symptom in the newly born. Its absence never permits the diagnosis of sepsis to be excluded. It is easy to understand that debilitated and premature children do not become feverish, when one considers that the subnormal temperatures existing in such children are often not raised much above the low level when heat is applied. The heat-regulating centres also react little to septic poisons. But the absence of fever is not a peculiarity of children disposed to subnormal temperatures; relatively strong newborn infants also show sometimes, particularly in genuine toxæmia, and also in pyæmic affections, either no rise in temperature or only a very slight and transient fever, and this may easily pass unnoticed. Sometimes the affection begins with fever which disappears after a relatively short time, or has an irregular course. On the other hand a feverish course of the disease may also occur with sepsis of the newly born. The temperatures may be very high and mount to 104° F. and beyond. The fever may be intermittent, remittent or continuous. Continuous fever or that returning after a short time, is also an important diagnostic sign of infection in the newborn. Rigors never occur in small infants, but they show a remarkable disposition to symptoms of collapse.

Symptoms on the part of the central nervous system may be due to meningitic or encephalitic complications, or to oedema and hyperæmia of the meninges, and last but not least, to toxic influences. Thus, one occasionally observes signs of cerebral irritation, states and excitement, screaming, spasms, hypertonia, and tremor; but as a rule these signs are infrequent, lifelessness and stupor are decidedly more frequent.

The changes in the type of respiration which are occasionally observed are probably of cerebral origin, such as acceleration and pronounced depth of the respirations, sometimes a marked dyspnoea. In many cases vomiting may also be considered as a toxic cerebral symptom.

As with all parenteral infections of infants, secondary diarrhoea may also occur in the course of non-enterogenous sepsis of the newborn. Exanthemata must be regarded as very characteristic symptoms of sepsis, though only found in a relatively small number of cases, sometimes appearing as a scarlatiniform or morbilliform erythema, sometimes as a pustular eruption. In particular haemorrhagic exanthemata are almost exclusively of septic origin; efflorescences resembling purpura, or large extravasations may be found, and sometimes the very characteristic haemorrhagic pemphigus.

How far the haemorrhagic symptoms in other organs may be of septic origin, has already been discussed. The haemorrhages which are found in the course of a septic affection, not only in the skin, but also in the mucous membranes and internal organs, are due to toxic injury of the walls of the vessels and must therefore be counted among the general symptoms. If in an indefinite condition which resembles sepsis, haemorrhages from the navel, skin, nose and hæmatemesis, &c., occur, then these are ominous symptoms and serve to confirm the diagnosis.

According to reports from the pre-septic period (Ritter, Eysela) in haemorrhagic sepsis umbilical haemorrhages appear to stand in the foreground of the clinical picture. In 1873 Ritter reported 100 cases of "haemorrhage in earliest infancy" in which the navel escaped in only 38 cases; 97 cases concerned the navel alone; in 25 cases umbilical haemorrhage was combined with others (from mouth, stomach, intestine, skin, connective tissue of the eyelids, external ear, nose, bladder, female genitals). The haemorrhages which in a large number of cases were doubtless accompanying symptoms of jaundic affections, were described by Ritter as the expression of "temporary hæmophilia of toxic origin." They generally appeared during the second week of life. In this respect they show a very essential difference from "idiopathic" haemorrhagic affections of the first few days of life.

The course of septic infection is extremely multiform. Apart from those violent cases which lead to death within a very short space of time, there are sub-acute and chronic forms which may last for weeks. Sometimes the disease pursues its course in the most alarming manner, with high fever and all the described general symptoms; in other cases the form of disease is so indefinite that one may finally be in doubt whether a general infection is actually present, or merely a general constitutional weakness, the condition resulting from underfeeding, or the onset of nutritional disturbance. These forms of the disease without symptoms are relatively frequent in premature children. The diagnosis of "sepsis," particularly when there is no clinical evidence of a local affection, is often just as difficult to confirm with any degree of certainty as it is to exclude. Our clinical methods of research very seldom give decisive information. The unreliability of the temperature has already been pointed out. The presence of an acute tumour of the spleen is, in fact,

diagnostically a most significant symptom, but one should guard against accepting every palpable spleen as a septic tumour of the spleen. If more albumin is found in the urine than should correspond to the age of the child, a general infection may be the cause; but it may also be the result of a non-bacterial intoxication; possibly also of underfeeding. The morphological examination of the blood does not as a rule provide any definite information, as leucocytosis is generally absent in sepsis of the newly born. Diminution in the coagulability of the blood is also no pathognomonic symptom of sepsis if one is not disposed to associate the haemorrhagic affections with it. The most definite information may, of course, be expected from the bacteriological examination of the blood; this has in fact often made the establishment of the diagnosis possible. But quite apart from toxæmia, in which no bacteria circulate in the blood—the blood, if the result is to be of any use, must be obtained under such aseptic conditions that its general use for diagnostic purposes meets with considerable difficulties. And therefore we must often be satisfied with a diagnosis of probability.

The prognosis of septic affections is extremely serious, at least, if the sepsis can be definitely diagnosed from the clinical aspect. The injury to the whole organism is, in such cases, so considerable, that complete recovery is no longer possible; not even if it is possible to remove the septic micro-organisms from the body. On the other hand, there is no doubt that mild or slow infection can be successfully dealt with. It is quite possible that some disturbance in nutrition or growth, at a later period of infancy, apparently due to constitutional weakness, or some marasmic or atrophic state, has its origin in an unknown septic infection of the newborn period, whether it be due to "chronic sepsis" or to some inferiority and deficient organic resistance resulting from the past infection.

The treatment of manifest septic affections is not promising. No very satisfactory results have been attained up to date from the injection of antisera. There is also considerable scepticism as to the effect of other methods against infection (collargol, electrargol). As they are certainly harmless they may be tried, in default of other more efficacious methods, either intravenously or subcutaneously. Repeated infusions of saline are much to be recommended to check exsiccation; possibly the effect is good in washing out poisonous substances from the body. For the same reason an abundant supply of liquid should be administered per os; this may be somewhat difficult owing to the unwillingness of the affected children to drink. Collapse should be treated by the usual stimulants (injection of *öl. camph.* 0.05 gm., *digalen* $\frac{1}{2}$ c.c.m., *cafein sodium salicylate* 0.05 gm., &c.); mustard baths or compresses have also a good symptomatic effect. In high fever cool packs generally quickly reduce the temperature.

To feed a septically affected child with anything but mother's milk, may be *a priori* considered as useless. As in these cases the amounts drunk are always so slight, a relatively poorly secreting

breast will generally be able to give sufficient. Owing to the feeble sucking power of the child the milk should be drawn off and administered by means of the bottle or spoon.

Prophylaxis of Sepsis. Care of the Newborn.

Although on the appearance of clinical symptoms of septic disease, the fight should not straightway be abandoned, in spite of the gloomy prognosis, yet our efforts to cure sepsis are, up to date, not very promising. Our first duty, however, by no means lies in this direction. Nowadays we know the means and ways of protecting the newborn child to a very large extent from these previously devastating septic diseases. Though cases of sepsis still occur, the reason is probably partly that our prophylactic methods are still somewhat limited (infections ante and intra partum). On the other hand, it cannot be denied that in the case of the newborn sufficient precaution is not invariably used, though it is so necessary, in consideration of its enormous hygienic importance. Even if it lies beyond our power to protect the individual from that which was established during his foetal existence and produced by his constitution, from that which he inherited from his parents and forefathers, yet we are in a position to protect the newborn child from the very day of its birth from those dangers which accompany extra-uterine life. Of the three groups of disturbances of nutrition of the infantile period (using a more comprehensive term we may also speak of "disturbances of health," viz., disturbances due to the constitution, feeding and infection), we need only consider the first as unavoidable; both the others we can check; we must create for the child the most propitious conditions of feeding, using all our efforts, under the circumstances, to promote feeding with mothers' milk; by means of asepsis and cleanliness we must keep it free from any avoidable infection. Sooner or later, everybody, live he ever so hygienically, exposes his health to serious dangers; the problem of the care of the infant is to keep at a distance as far as possible all those dangers, at least during the first period of life. The healthier he is when he meets them later for the first time, the easier can he resist them. Clinical experience shows that with healthy children the resistance to alimentary and infective injuries during the first year tends to strengthen with increasing age, and that the prognosis of many morbid conditions, which is most serious during the first three months, improves during the next three or six months. The newborn child, for whom every physiological stimulus represents something new and unaccustomed, and who under entirely normal conditions shows some backwardness in his ordinary functions, is particularly sensitive and vulnerable with regard to many a cause of disease.

Apart from the general rules applicable to the care of the child during infancy, special regulations are necessary for the newborn child. There is a gap in the knowledge of many modern infants'

nurses, in that they possess no practical experience of the characteristics of the newborn and the special knowledge required in the nursing. Apart from the treatment of the umbilical cord and wound (see p. 418) comes foremost the technique of feeding during the first few days of suckling, which with difficulties in feeding demands great experience and the knowledge of many special devices, such as can only be gained by practice. How important is the work of the nurse attending the mother during the period of suckling, has already been discussed. A further and not less important part of the care of the newly born consists in the prophylactic measures which are necessary for the prevention of infection during the mother's confinement.

If we consider the necessary prophylactic measures in lying-in institutions, the ideal arrangement would be the absolute separation between the nursing of the child and the woman and the utmost possible separation of the children from the lying-in women. The children should be kept in their own room and only brought into the lying-in room for their feeds; they should have their own staff of nurses, who have nothing to do with the women except the cleansing of their breasts. But such an organization, corresponding to all the modern demands of hygiene, and so beneficial to mother and child, is unfortunately not found in all lying-in institutions. Even in large, modern-equipped women's clinics it is often still the custom, that the newborn children are kept in the lying-in rooms and in the immediate vicinity of the women, and that the same staff (often insufficient in quantity) attends to the mothers and children. It must not be suppressed that such old-fashioned arrangements, adhered to for reasons of economy, are retained at the cost of order and hygiene, and that unfortunately many septic diseases of the children must be primarily ascribed to them. How desirable it would be for the latest reforms to be introduced, may be gathered from the wonderful results which the asepsis of modern midwifery has also brought for the children. Infections occurring during and immediately after birth have been, on the whole, reduced to a minimum; infections of the child from instruments, linen, bandages, &c., only occur nowadays through gross violation of obstetric regulations. Also the frequency of severe infections of the children, arising from infected genital secretion, is reduced as far as possible, owing to the prophylactic measures, in the examination of pregnant women, &c. But just in this respect further measures of precaution are very necessary. The lochial secretion always contains bacteria, which even in clinically healthy mothers are of a pathological nature, in any case for the newly born. The preservation of the child from this important source of infection in lying-in rooms must be taken into consideration. Soiled utensils and linen must be placed at once in closed receptacles or immediately removed from the room in order to prevent the drying of the infective secretion and the infection of the air resulting thereby. But still more dangerous than the latter is the transmission of infective germs by the hands of the mother

and nurse, who are bound to come into contact with infective material, in spite of the greatest cleanliness. For this reason the mother, at least, should handle the child as little as possible; inasmuch as this cannot be avoided, she should be informed on the subject, so that she should touch the child and her nipples only with her hands well cleaned. It has already been mentioned that the breasts should be thoroughly washed daily, that they should be covered with a clean cloth, or piece of muslin or, in any case, with perfectly clean body linen. Everything else that comes into contact with the child's mouth (breast-shields, sucker, &c.) should be boiled daily or sterilized in steam and retained between whites in boiled water or boracic solution, or kept in a clean cloth.

In many lying-in institutions, separation of the children from the mothers is not carried out, but at least the nursing of the women and children is undertaken by a separate staff. Where this is not the case, the hygienic demands made on the nurses should be particularly strict. But unfortunately in this respect the demands and desires are far from fulfilment.

In the guide to the nursing of infants by Pescatore-Langstein the following are the principal orders given to the nurses:—

(1) Never touch two children in succession without first thoroughly washing the hands. In other words: after contact with one child your steps should lead you mechanically and with automatic regularity to the washing basin.

(2) Every utensil which, either directly or indirectly, has been brought into contact with one child, should only be used for this child, or should be thoroughly disinfected before further use.

Though the second of these "golden rules on the nursing of the infant in institutions" is carried out in modern lying-in institutions, the first meets with many obstacles owing to too much pressure being laid on the staff. If in the course of twenty-four hours a child is only changed six times, the nurse, if she has to look after the children herself, would have to wash her hands at least 120 times a day, with a room containing twenty lying-in patients; if she also has to nurse the lying-in women, even with the help of an assistant, the necessary times of washing her hands would not be underestimated at 200. It is simply impossible that cleanliness in such a case could be so strictly adhered to.

It, in spite of these unfortunate conditions, septic infections relatively rarely occur and are only exceptionally of a severe nature (this, as previously mentioned, is due to the asepsis during confinement and the cleanliness of the room; the opportunity of infection is relatively slight. But if infectious diseases, even in modern lying-in institutions, are not amongst the rare exceptions, it is because the nursing of the child has not reached the level which is so necessary in all infant institutions. It is particularly to be regretted in consideration of the danger of infection just during the newborn period. And this is only a question of money: as soon as the number of nurses is increased and the modern hygienic demands

(certainly not exaggerated) for the care of the infant are fulfilled, as soon as a sufficient quantity of linen is provided for the children, the nursing of the newborn (often many decades behind the times) in lying-in institutions will show such success, that all the children will leave the institution in perfectly healthy condition.

The same rules apply essentially to the nursing of the newborn in private houses. As one nurse is seldom provided for the infant alone, care must be taken that the midwife, responsible for the nursing of mother and child, should first attend to the child and before coming into contact with it, should thoroughly wash her hands.

The newborn child, if not bathed, should at least be thoroughly washed every day (see p. 421). In order to prevent an intertrigo occurring, frequent changing of the child into clean dry napkins is very necessary. Powdering with a thin layer of powdered asbestos or any infant powder, is not necessary but harmless, if the skin is intact.

With regard to the clothing of the newborn the same rules apply as for the infant period generally: above the triangular-folded napkin, covering the skin, a smaller or square-folded napkin should be placed together with a piece of impermeable material or mackintosh, which must not be too big so that it encircles the body. Finally the child should be wrapped up in soft flannel, as loosely as possible, though firmly. The upper part of the body should be clothed with a tiny shirt and jacket. With regard to pillows and swaddling bands, modern paedetrists are against their use; but for the newborn child they will probably be allowed, though the usual covering and head pillow are sufficient. It goes without saying that the child must not be strapped in tightly, that the easily flexed legs should not be forcibly extended and that the arms, as a rule, should not also be encased; owing to the thermolability of the young infant the warm swathing of the swaddling bands is not only beneficial for premature and weakly children, but also for full-time healthy ones. For weakly children, in spite of the swaddling bands, hot bottles should be placed in the bed for the first few days, in order to keep the body temperature at a normal level.

It would seem almost superfluous to point out that swaddling bands should be washable, and like the rest of the linen, white, in order to preserve the strictest cleanliness. But unfortunately, this is not always the case. One sees with astonishment that even in the most modernly equipped hospitals the children are wrapped in the gaudiest coloured coverings, and not even in accordance with the most modest demands of cleanliness—and merely because the economical hospital authorities do not supply any linen, beyond napkins, for the newborn children.

Owing to the warmth of the clothing the usual temperature of the room need not exceed 59° F. A moderately tempered atmosphere is always advantageous for the child. By means of thorough ventilation care must therefore be taken to keep the air of the room

always fresh and pure. Besides air, light should always be allowed to enter the room in which the newborn child is lying (see p. 75).

It is therefore the duty of the medical attendant and nurse to see that the infant lives in a hygienic atmosphere from the very first day of its birth, and one containing no pathogenic germs injurious to the child. In this respect puerperal affections of the mother are liable to be feared. With conscientious nursing the child can be protected from these with almost positive certainty, without being removed from the mother; in such cases suckling is seldom accompanied by any disadvantages. Far more dangerous are the affections of the respiratory organs, particularly pulmonary tuberculosis, which are air-borne. It is of the greatest importance that the persons in the vicinity of the newborn child should be healthy in this respect.

Even when the greatest precautions are taken with newborn children, with the best of motives the precautionary measures are often extremely distorted. One may be afraid of the child catching cold and wrap it up in a most exaggerated manner, others carefully guard it against fresh air and keep it sheltered in the dark in order to preserve its eyes—all these precautions being not only superfluous, but sometimes directly injurious. On the other hand they may overlook the fact that an ordinary cold or slight catarrh in an adult may be injurious to a young infant, and how dangerous too close contact with the lying-in woman may be.

Enlightenment on the rules in nursing the newly born is one of the most important tasks of hygiene. Prophylaxis, the principal branch of therapy, must begin on the very first day of life.

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